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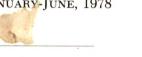
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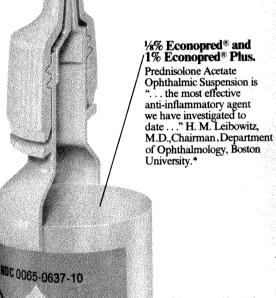
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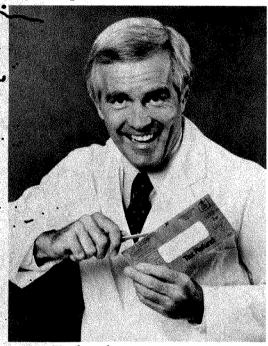
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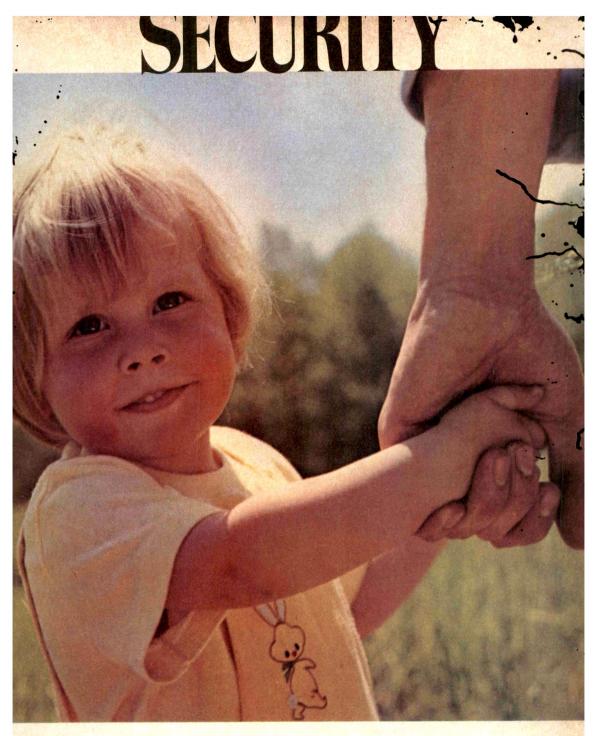
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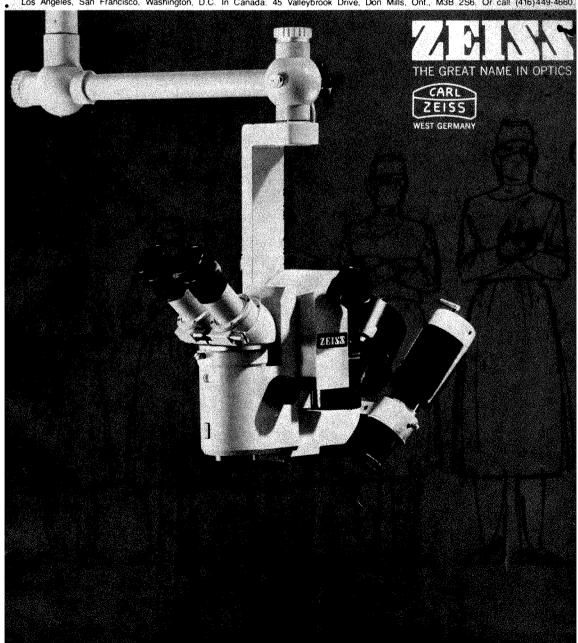
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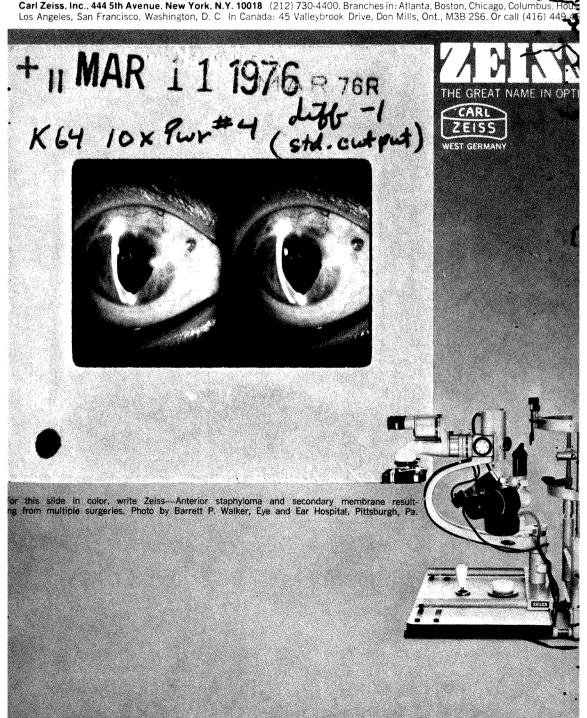
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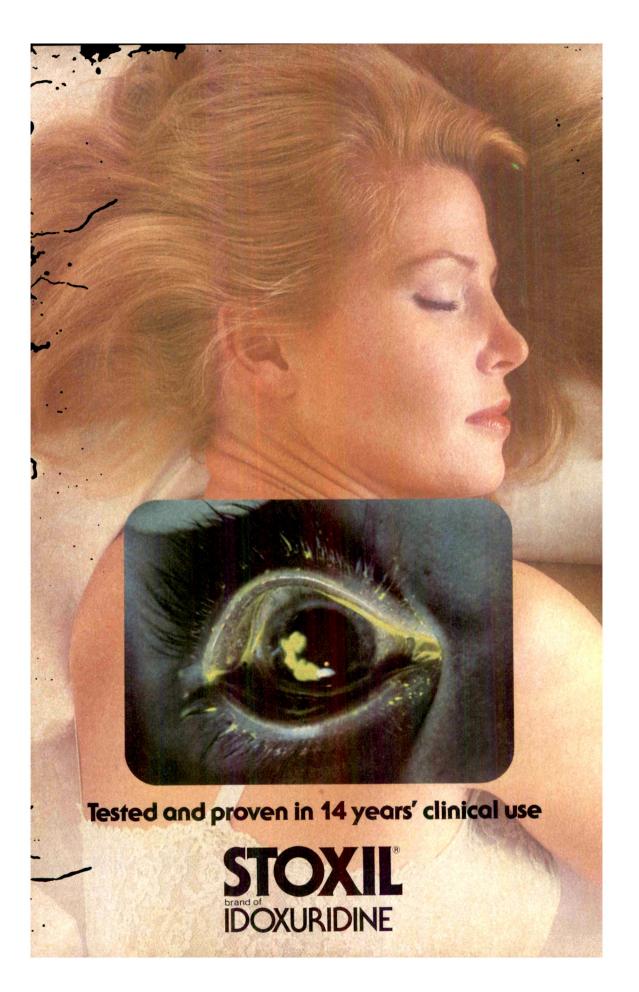
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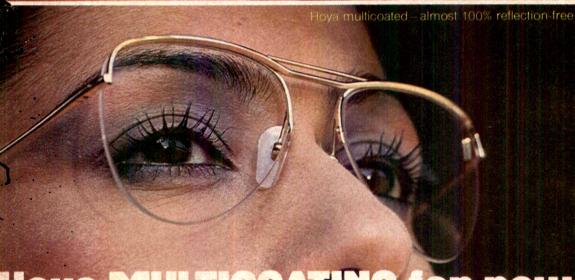
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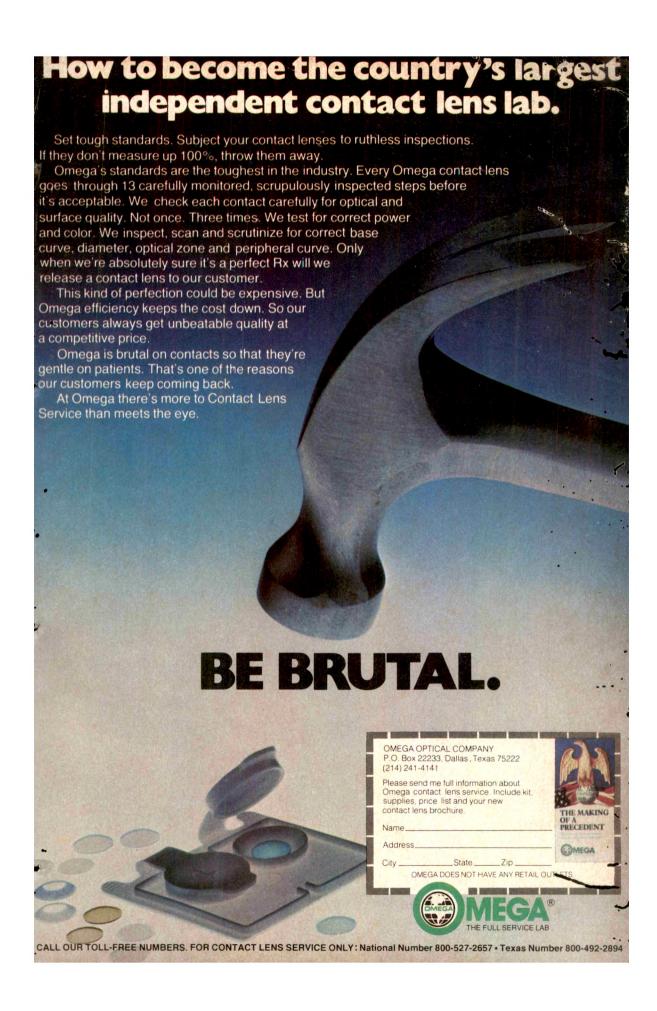
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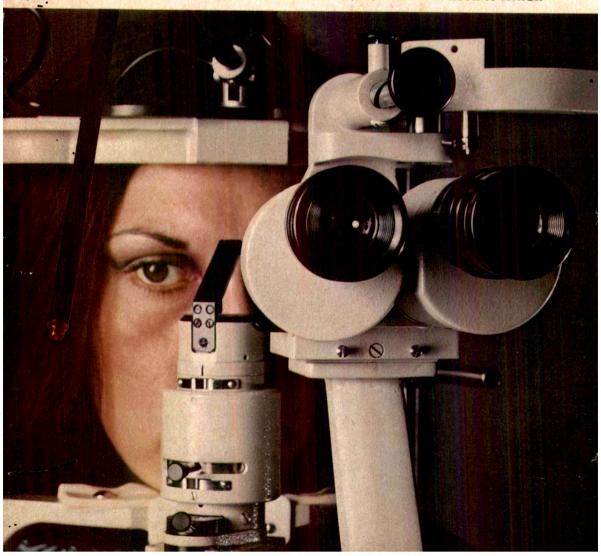
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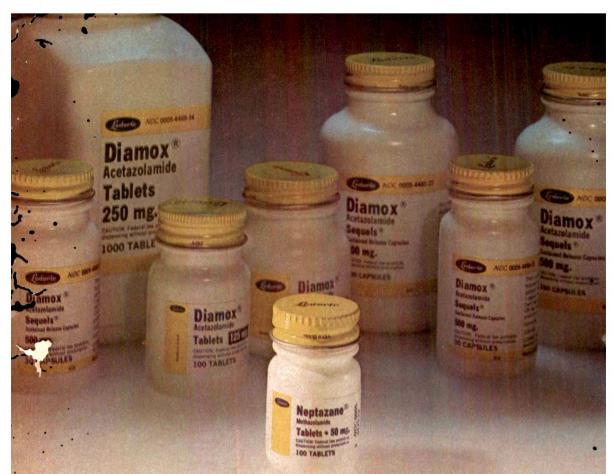
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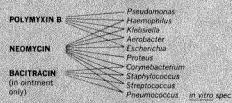
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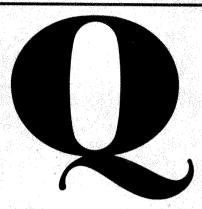
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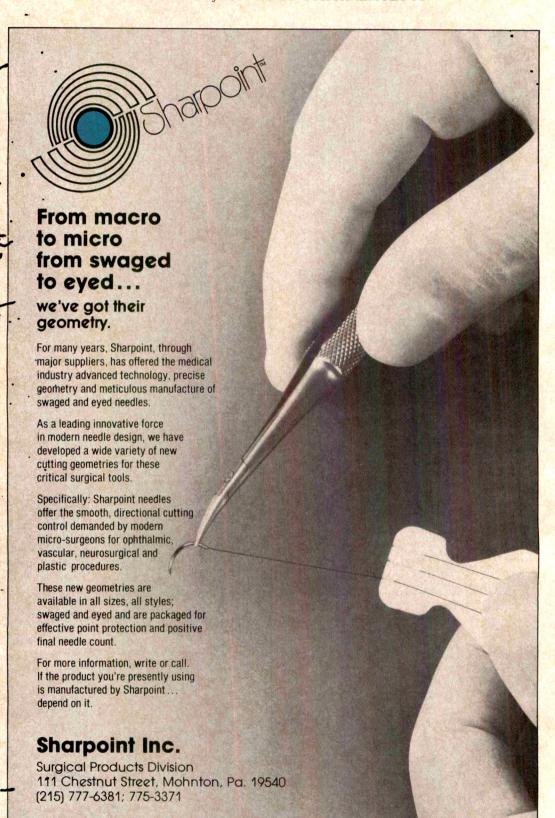
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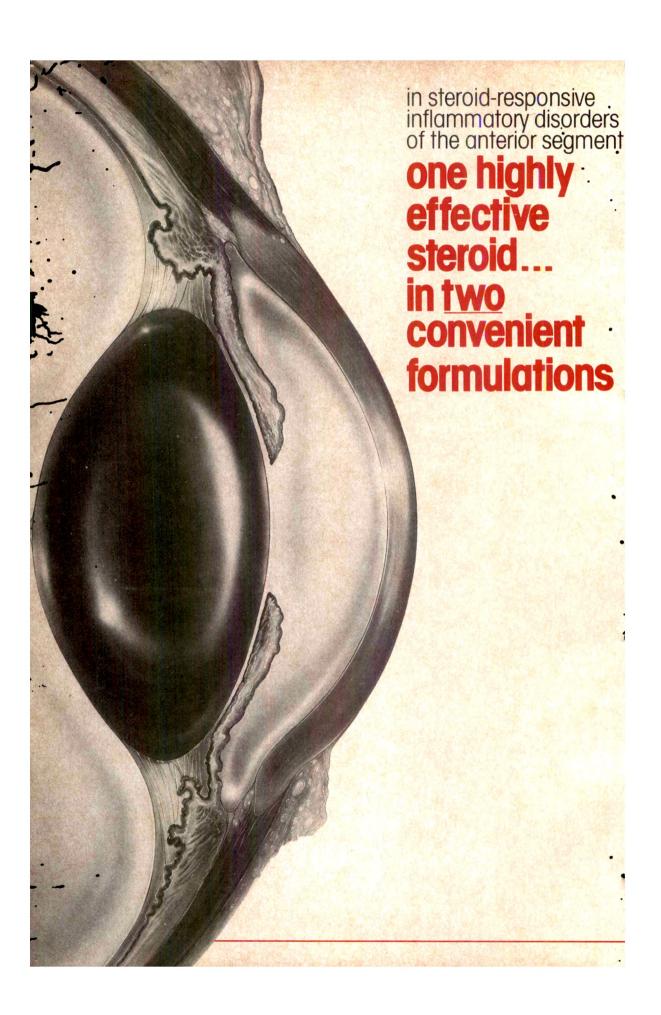
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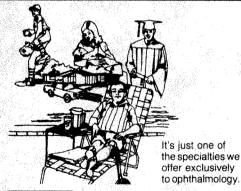
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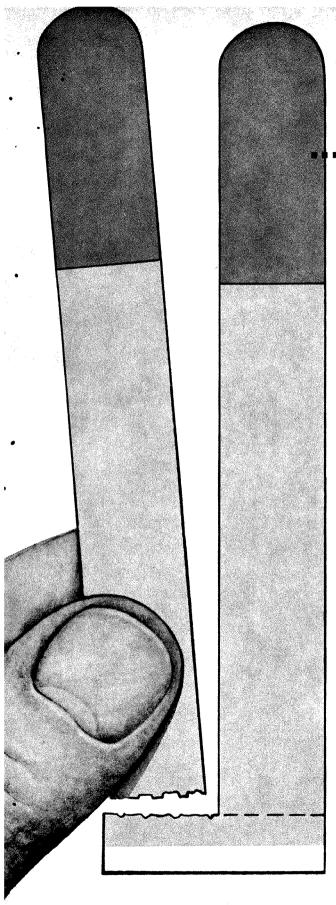
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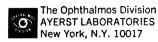


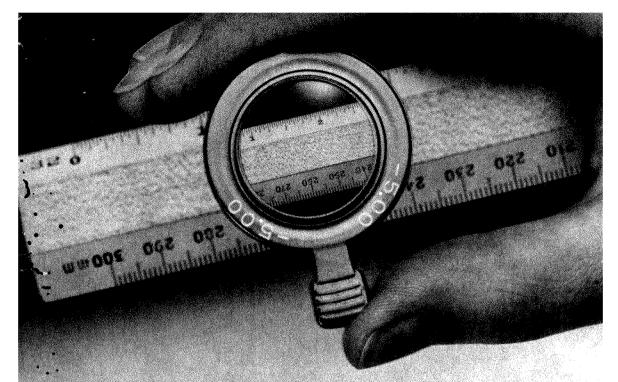
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Curriculum will include live and closed circuit observation of surgery, practice surgery, formal and informal discussion, observation of immediate and long term postoperative follow-up.

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Guest Speakers Frederick C. Blodi, M.D. Lorenz Zimmerman, M.D.

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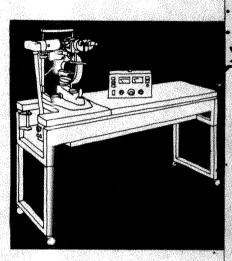
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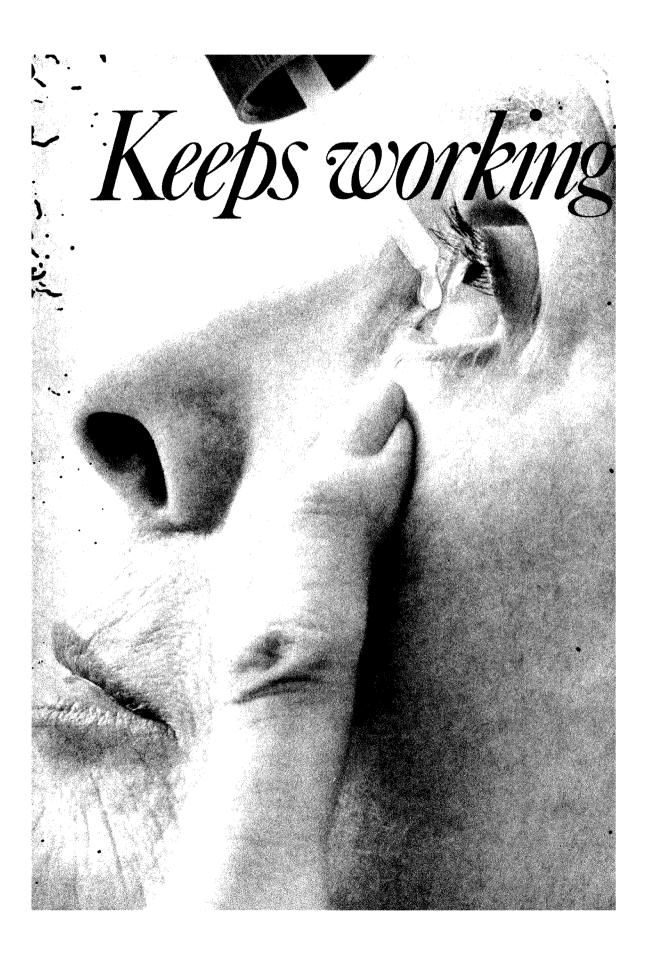
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Phospholine Iodide (echothiophate iodide for ophthalmic solution)

See next page for prescribing information.

Phospholine Iodid (echothiophate iodide) in the management of chronic 'simple glaucoma or glaucoma secondary to aphakia

BRIEF SUMMARY
(For full prescribing information, see package circular)
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(ÉCHOTHIOPHATE IODIDE FOR OPHTHALMIC SOLUTION)

PHOSPHOLINE IODIDE is a long-acting cholinesterase inhib-

Indications: Glaucoma—Chronic open angle glaucor Subacute or chronic angle closure glaucoma after indectomy or where surgery is refused or contraindicated. Certain non-uveitic secondary types of glaucoma, especially glaucoma following cataract surgery.

Accommodative esotropia - Concornitant esotropias with a significant accommodative component.

Contraindications: 1. Active useal inflammation.

2. Most cases of angle-closure glaucoma, due to the possibility of increasing angle block

3. Hypersensitivity to the active or inactive ingredients.

Warnings: 1 Use in Pregnancy. Safe use of anticholinesterase medications during pregnancy has not been established, nor has the absence of adverse effects on the fetus or on the respira-

nas the absence of adverse effects on the tetus or on the respiration of the neonate.

2. Succinylcholine should be administered only with great caution, if at all, prior to or during general anesthesia to patients receiving anticholinesterase medication because of possible respiratory or cardiovascular collapse.

3. Caution should be observed in treating glaucoma with PHOSPHOLINE IODIDE in patients who are at the same time.

undergoing treatment with systemic articholinesterase medica-tions for myasthenia gravis, because of possible adverse additive

Precautions: 1. Gonioscopy is recommended prior to initiation

2. Where there is a quiescent uveitis or a history of this condition, anticholinesterase therapy should be avoided or used cautiously because of the intense and persistent miosis and

ciliary muscle contraction that may occur.

3. While systemic effects are intrequent, proper use of the drug requires digital compression of the hasolacrimal ducts for a minute or two following instillation to minimize drainage into the nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation.

4. Temporary discontinuance of medication is necessary if

salivation, urinary incontinence, diarrhea, profuse sweating, muscle weakness, respiratory difficulties, or cardiac irregularities

Patients receiving PHOSPHOLINE IODIDE who are ex osed to carbamate or organophosphate type insecticides and pesticides (professional gardeners, farmers, workers in plants manufacturing or formulating such products, etc.) should be warned of the additive systemic effects possible from absorption of the pesticide through the respiratory tract or skin. During periods of exposure to such pesticides, the wearing of respiratory masks, and frequent washing and clothing changes may be advisable. advisable

6. Anticholinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulcer, pronounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects.

7. Anticholinesterase drugs should be employed prior to ophthalmic surgery only as a considered risk because of the possible occurrence of hyphema.

8. PHOSPHOLINE IODIDE (echlothiophate iodide) should be used with great caution, if at all, where there is a prior history of retinal detachment.

8 PHOSPHOLINE IODIDE (echothiophate iodide) should be used with great caution if at all, where there is a prior history of retinal detachment.

Adverse Reactions: 1 Although the relationship, if any, of retinal detachment to the administration of PHOSPHOLINE IODIDE has not been established, retinal detachment has been reported in a few cases during the use of PHOSPHOLINE IODIDE in adult patients without a previous history of this disorder.

2 Stinging, burning, lacrimation, lid muscle twitching, conjunctival and clilary redness, browache, induced myopia with visual blurring may occur.

3. Activation of latent irrits or uveitis may occur.

4. Iris cysts may form, and if treatment is continued, may enlarge and obscure vision. This occurrence is more frequent in children. The cysts usually shrink ispon discontinuance of the medication, reduction in strength of the drops or frequency of instillation. Rarely, they may rupture or break free into the aqueous. Regular examinations are advisable when the drug is being prescribed for the treatment of accommodative esotropia.

5. Prolonged use may cause conjunctival thickening, obstruction of nasolacrimal canals.

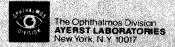
6. Lens opacities occurring in patients under treatment for glaucoma with PHOSPHOLINE (ICDIDE have been reported and similar changes have been produced experimentally in normal monkeys. Routine examinations should accompany clinical use of the drug.

7. Paradoxical increase in intraocular pressure may follow anticholinesterase instillation. This may be alleviated by prescribing a sympathomimetic mydriatic such as phenylephrine.

Overdosage: Antidotes are atropine. 2 mg parenterally.

PROTOPAM* CHL.ORIDE (pralidoxime chloride). 25 mg per kg intravenously, artificial respiration should be given if necessary.

How Supplied: Four potencies are available 1.5 mg package for dispensing 0.03% solution. 3.0 mg package for 0.06% solution: 6.25 mg package for o.03% solution. 3.0 mg package for 0.06% solution: 6.25 mg package for o.05% solution: acid and existoated sodium p



Announcement of the

EIGHTEENTH ANNUAL INSTRUCTIONAL COURSE IN CONTACT LENS FITTING BY THE OPHTHALMOLOGIST

March 9-10-11, 1978 • NEW ORLEANS

THE RUDOLPH ELLENDER MEDICAL FOUNDATION

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Tuition: \$195.00

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Special Guest Lecturer: **Jack R. Anderson, M.D.**, New Orleans Saturday, March 11, 1978—Treatment of Aging Eyelids

Additional information: Second Annual OphthalmoCryosurgical Seminar will follow the above course—March 11 & 12, 1978

For further information contact:

Jos. A. Baldone, M.D.

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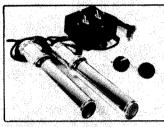
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Each cc contains: Aerosporin* brand Polymyxin B Sulfate 5,000 Units, neomycin sulfate 2.5 mg (equivalent to 1,75 mg neomycin base); gramicidin 0,025 mg. Vehicle contains alcohol 0.5%, thimerosal (preservative) 0,001% and the inactive ingredients propylene glycol, polyoxyethylene polyoxypropylene compound, sodium chloride and ourified water.

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Brief Disclosure below applies to the solution and pintment.

tion and ointment.
INDICATIONS: For the short-term treatment of superficial external ocular infections caused by organisms susceptible to one or more of the antibiotics.

CONTRAINDICATIONS:

Contraindicated in those persons who have shown sensitivity to any of the components

WARNINGS:

Prolonged use may result in overgrowth of nonsusceptible organisms. Ophthalmic Ointment may retard corneal healing.

PRECAUTIONS:

Culture and susceptibility testing should be performed during treatment.

Allergic cross-reactions may occur which

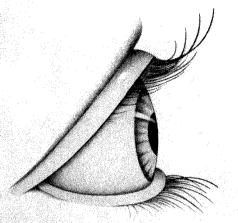
Allergic cross-reactions may occur which could prevent the use of any or all of the following antibiotics for the treatment of future infections: kanamycin, paromomycin, streptomycin, and possibly gentamicin.

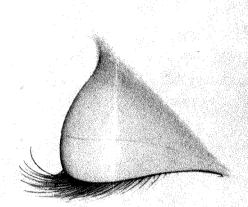
ADVERSE REACTIONS:

Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Complete literature available on request from Professional Services Dept. PML.



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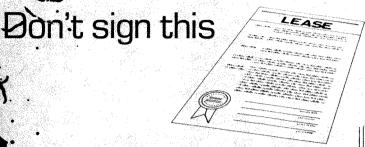




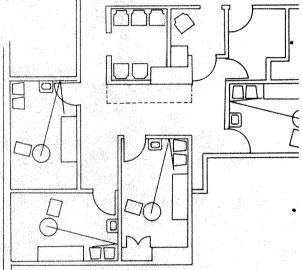
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See adjacent page for brief prescribing information

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Richard Troutman, M.D. on Anterior Segment Surgery

Jack Harstein, M.D. on Ultrasound Cataract Surgery and Intraocular Lenses

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MARCH 4 - 8, 1978 ◆ HYATT REGENCY ◆ NEW ORLEANS, LOUISIANA

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Phacoemulsification: Indications, Contraindications, Results

Phaco.: Past Present and Future Small Incision Intraocular Lenses

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Kvoto, Japan — May 14-20, 1978

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Announcement of

SECOND ANNUAL OPHTHALMOCRYOSURGICAL SEMINAR

(To be presented in conjunction with the 4th Annual DermatoCryosurgical Seminar and to be proceeded [March 9-11, 1978] by the 18th Annual Instructional Course in Contact Lens Fitting by the Ophthalmologist and followed by the 1st Annual American Coilege of Cryosurgery Program, March 13, 1978)

Sponsored by RUDOLPH ELLENDER MEDICAL FOUNDATION

A.M.A.-approved Continuing Medical Education Program (8 hours Credit toward Physician's Recognition Award Category 1)

Saturday, March 11, 1978 and Sunday, March 12, 1978 (2:00 til 6:00 p.m.) (8:00 a.m. til noon)

PLACE:

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TOPICS:

Cryobiology and Cryosurgery of the Eye and Adnexa

TUITION:

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For further information contact:

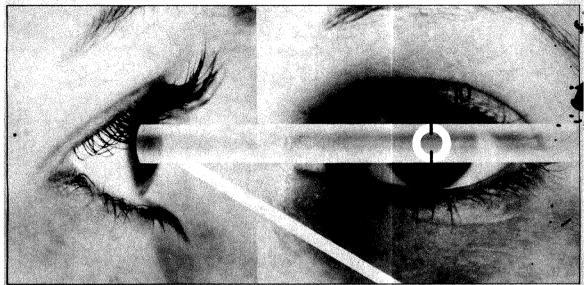
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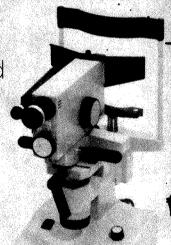
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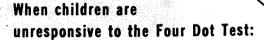
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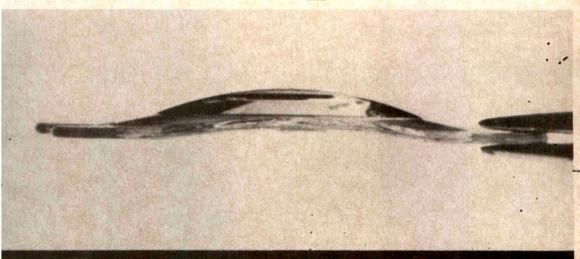
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Saturday, May 20: Entropion, Ectropion, Orbital Fractures, Lid & Orbital Tumors

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Precautions: 1 Gonioscopy is recommended prior to initiation of therapy.

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occur.

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6. Anticholinesterase drugs should be used with extreme o. Anticnolinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulcer, pro-nounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects.
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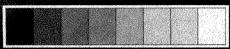
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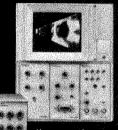
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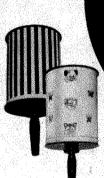
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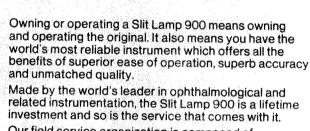
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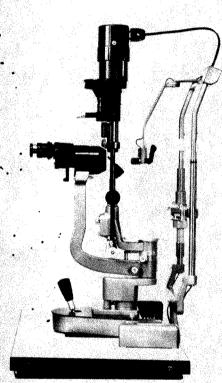


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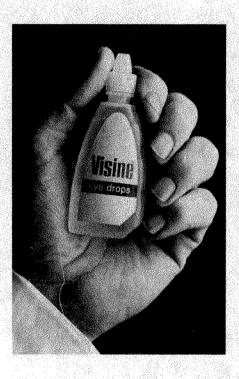
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DRUSEN OF THE OPTIC DISK AND ABERRANT AXOPLASMIC TRANSPORT

THE XXXIV EDWARD JACKSON MEMORIAL LECTURE

WILLIAM H. SPENCER, M.D.

San Francisco, California

The Edward Jackson lectureship perpetuates the memory of a distinguished physician; we remember him chiefly for his contributions to the field of refraction and for his editorial activities. But he was an effective teacher, an enthusiastic supporter of scientific societies, and an innovator of postgraduate educational programs as well. Thus, it is particularly fitting to present the Jackson Lecture before this Academy, both because he contributed so much to it and because it is at the forefront of ophthalmic education. I am profoundly grateful for the honor of presenting the XXXIV Edward Jackson Lecture.

Since the original histologic observation of drusen of the optic disk 119 years ago, numerous publications have delineated their clinical manifestations, but no one has satisfactorily spelled out their cause. I intend to discuss the proposition that alterations in the dynamics of axoplasmic transport cause drusen of the disk. The material and views I shall present were derived in collaboration with my colleague, William F. Hoyt. In 1962 Seitz and Kersting² suggested that drusen of the disk may be the product of long-term pathologic alterations in the retinal nerve fibers. In 1968, Seitz³ proposed this idea again and concluded from a series of histochemical studies that drusen originate from axoplasmic derivatives of disintegrating nerve fibers. Seitz also made the important observation that drusen form by a slow degenerative process rather than a rapid one.

Clinical observations—Before discussing these two observations further, I would like to explain the settings in which drusen develop, particularly those that occur on a familial basis and those associated with retinitis pigmentosa.

The familial type is typically bilateral and appears to be inherited as an irregular dominant4 (Fig. 1). Initially, drusen were considered static, but it has become increasingly apparent that they evolve slowly, often requiring decades to develop fully. Early in life the disks appear "full" and physiologic cupping is absent; therefore, the processes leading to drusen occasionally mimic papilledema. Later, the disk acquires a tan, vellow, or straw color, and eventually yellow-white spherical glistening structures that vary in number and size become visible. Initially, one can detect these structures at the margin of the elevation beneath the surface and can see them best by indirect illumination. In adult life, the disk sur-

From the Department of Ophthalmology, Pacific Medical Center, San Francisco, California. Presented before the 82nd annual meeting of the American Academy of Ophthalmology and Otolaryngology, Dallas, Texas, Oct. 6, 1977.

Reprint requests to William H. Spencer, M.D., Department of Ophthalmology, Pacific Medical Center, 2340 Clay St., San Francisco, CA 94115.



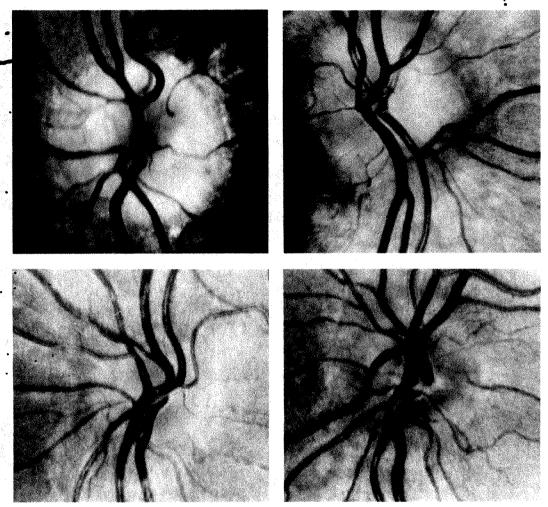


Fig. 1 (Spencer). Familial drusen of the disk. Top left, Mother. Top right and bottom left and right, Three children. All of the disks lack physiologic cups. The drusen are more apparent in the mother.

face appears studded with refractile bodies, particularly near the margins.

Associated visual field defects include generalized depression, relative arcuate scotomas, enlargement of the blind spot, and concentric constriction. Eyes we have observed ophthalmoscopically for many years have exhibited slowly progressive atrophy of the nerve fiber layer. Several reports even described blindness associated with drusen.⁵⁻⁷

The visual field defects do not corre-

spond to the position of the drusen; this has puzzled many. Drusen may be limited to one quadrant of the disk, yet the visual field shows concentric contraction and depression in all quadrants and the nerve fiber layer exhibits diffuse thinning as well.

One often sees anomalous branching of the major disk vessels in patients with drusen, as well as in family members with congenitally full disks.^{8,9} Occasionally, as the drusen evolve, intercurrent acute vascular events such as hemorrhage may take place. 10,11 These may take the form of splinter hemorrhages of the disk, vitreous, or subretinal hemorrhages.

Some studies report an abnormally small diameter of the nerve head.6.9 We attempted to corroborate this finding by reviewing our fundus photograph files and studying the appearance of the optic disk in patients we have seen clinically. However, because of the patients' refractive errors, variable degrees of magnification of the disk occur in the photographs. Furthermore, the scleral ring is often difficult to see because disk tissue and refractile bodies overlap its margin. Nevertheless, our impression is that the disks in patients with familial drusen are clinically smaller than normal, and none of the cases we reviewed had larger than normal disks.

Drusen in patients with retinitis pigmentosa have a different location and appearance. They can arise within the nerve head, but more often they lie just off the disk margin in the superficial retina¹²⁻¹⁵ (Fig. 2). The disk diameter appears normal. Unlike its appearance in

the familial group, the disk is not elevated. The disk tissue has a "waxy yellow" appearance and the lamina cribrosa is often concealed. The parapapillary drusen overlie blood vessels and do not enlarge, even when observed over a number of years (Fig. 3).

Histologic observations-Drusen are often seen in histologic sections as chance observations when they have not been clinically observed. In these instances they lie in the deeper aspects of the optice nerve head anterior to the lamina cribrosa and within the confines of the scleral ring (Fig. 4). Occasionally, the drusen are more superficial and overlie the disk margin where they are visible clinically: They are composed of calcified, concentrically laminated, globular aggregates that do not seem to form around cellular elements; they are, thus, unlike the corpora aranacea that develop around degener-. ated meningiothelial cells. A variety of, special stains have been utilized in an attempt to correlate the clinical and histologic evolution of drusen. These efforts have been hampered by the limits of magnification and resolution available

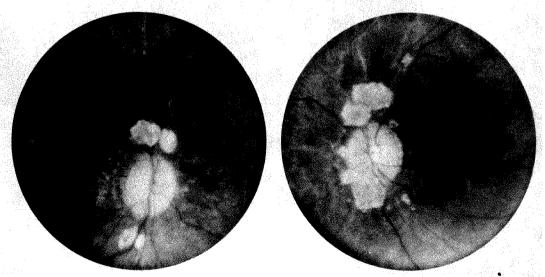


Fig. 2 (Spencer). Parapapillary drusen in retinitis pigmentosa. The drusen are located off the disk margin and in the superficial retina. Left, Right eye; and right, left eye of a 40-year-old man.

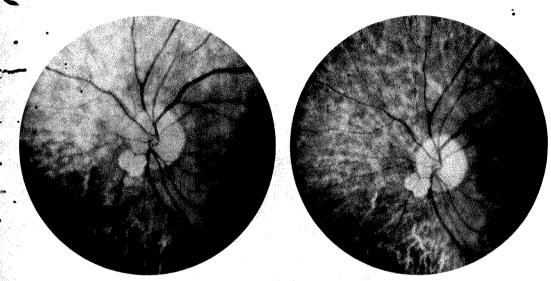


Fig. 3 (Spencer). Parapapillary drusen in retinitis pigmentosa. The drusen have not changed in appearance between 1965 (left) and 1977 (right) (From Robertson, D.M.).

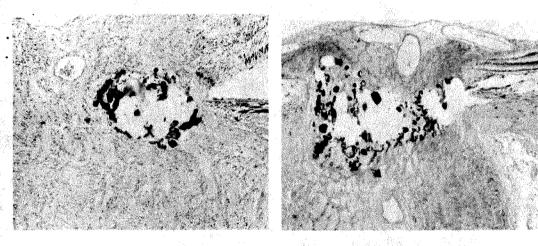
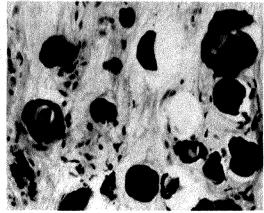


Fig. 4 (Spencer). Histologic appearance of typical drusen. Top left, Early drusen near disk edge. Top right, Moderately advanced drusen. Bottom right, Laminated globular calcified structures (hematoxylin and eosin; top left, ×4; top right, ×2.5; bottom right, ×82).



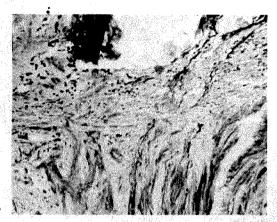


Fig. 5 (Spencer). Bodian stained section of right disk in an advanced case of bilateral drusen with almost total absence of axons. Eyes were obtained post mortem from a 74-year-old white woman with bilateral loss of light perception. The woman's son has bilateral drusen of the disk (×10) (From Zimmerman, L.E.⁵).

with the light microscope and the difficulty in obtaining suitable material for electron microscopy. Because the drusen are calcified, sectioning artifacts may occur. Decalcification before sectioning prevents this, but induces modifications in special stains. Nevertheless, special stains and careful sectioning have been valuable in providing histogenetic clues about drusen. In most specimens, stains for nerve fibers (Bodian) reveal diffuse depletion of axons, some terminating at the front of the drusen (Fig. 5). They also reveal a moderate amount of distortion and compacting of adjacent nerve fibers and glia as they are deflected around the drusen. To determine if the prelaminar axons contain accumulated mitochondria, we have utilized mitochondrial stains (Polak's modification of Del Rio Hortega's method, Altmann's aniline acid fuchsin, and methyl green). The results are uncertain. We observed collections of dots assumed to be mitochondria in thickened prelaminar axons in some sections, but the limits of resolution and the possibility of staining artifact prevent accurate interpretation. Calcified drusen also contain dense collections of black dots, but this appears to be an affinity of the stain of for calcium rather than mitochondria. We have not identified noncalcified deposits in the prelaminar tissue, which might be construed as the precalcified precursors of drusen. In this respect drusen of thedisk differ from drusen on Bruch's membrane where calcification occurs infrequently and seems to be deposited within pre-existent eosinophilic staining material.

We also observed an unusual nember of focal, amorphous, PAS-positive deposits resembling corpora amylacea in the prelaminar region of eyes containing drusen of the disk (Fig. 6). Similar deposits occur in degenerative diseases elsewhere in the central nervous system. They have been shown ultrastructurally to be composed of fibrillar material within abruptly distended axons. 16

Recently, Mark Tso, M.D., studied the ultrastructure of an optic nerve head containing drusen. He observed changes in prelaminar axons, glia, and vessels. His study is still in progress and he has had difficulty in differentiating primary from secondary changes. Despite this difficulty, he found prelaminar axonal swelling and disruption occurring not only in the area of the drusen, but in axons throughout the disk (Fig. 7).

Axoplasmic transport—After the pioneering report in 1948 by Weiss and Hiscoe, 17 studies established that proteins, sugars, subcellular organelles and other substances important in physiologic functions of the cell are transported along the axon at different rates of speed. Some substances move from the cell body toward the axon terminal (orthograde), while other substances are transported toward the cell body (retrograde). In slow orthograde transport, which has been likened to the flow of lava moving at 1 to 2 mm/day, much of the bulk of protein and other macromolecules and organelles are





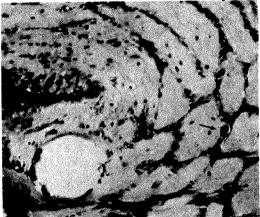
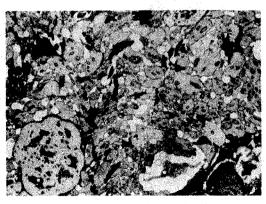


Fig. 6 (Spencer). Prelaminar focal amorphous deposits (arrows) in nerve fibers near drusen (left, hematoxylin and eosin, ×40; right, PAS, ×40).

utilized along the way, so that only a small portion reaches the axon terminal. Slow transport can be chronically blocked without arresting axonal conductivity.

On the other hand, fast orthograde transport moving at approximately 400 mm/day utilizes microtubules and neuro-filaments to carry materials destined for the axon terminal and depends on oxidative metabolism. With various pharmacologic agents and physical factors one can block fast orthograde transport to study

types of neuropathies. By using mild to moderate elevation of intraocular pressure in monkeys, investigators have blocked both slow and rapid axonal transport. 18,19 Ultrastructurally, the blockage occurs at the lamina cribrosa with striking distension of axons and pronounced accumulation of mitochondria and other axoplasmic particles (Fig. 8). Similar blockage of slow and rapid axonal transport at the lamina cribosa was produced by elevating pressure in the optic nerve vaginal sheaths²⁰; and, by lowering intra-



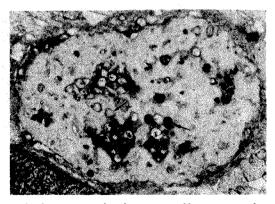


Fig. 7 (Spencer). Drusen of the disk. Electron micrograph of optic nerve head in region of lamina retinalis. Left, The axons show varying degrees of degenerative changes. Right, An enlarged axon in form of a cytoid body showing filamentous background and containing laminated bodies (black and white arrows) and dense bodies (black arrows) in the axoplasm (left, ×4,000; right, ×20,000) (From Tso, M.O.M.).

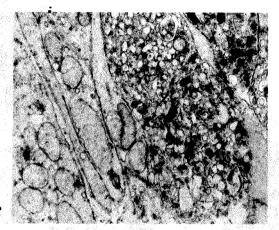


Fig. 8 (Spencer). Electron micrograph. Posterior lamina cribrosa Aotes monkey. Perfusion pressure of eye 45 mmHg. The distended axons contain accumulated axoplasmic particles and mitochondria (× 15,000) (From Hendrickson, A.).

ocular pressure, both slow and rapid transport was blocked.21-23 As a result of these studies, it has been suggested that damming the axoplasm as in such experimental situations partly aggravates a mechanical problem normally present in the nerve head. 22,24 Investigators have observed mitochondrial accumulations and axonal swellings at the lamina scleralis in the normal rhesus monkey eye.24 In studying serial cross sections of normal human and rhesus monkey optic nerve heads, Minckler, Tso, and Zimmerman²² discovered the narrowest aperture in the exit pathway of the optic nerve fibers lies at the level of Bruch's membrane. As long ago as 1911, Patton and Holmes²⁵ noted that in human papilledema the peripheral axons closest to the edge of Bruch's membrane are the first to swell and the most severely affected. Last year Minckler, Tso, and Zimmerman²² noted the most marked axonal swellings and accumulations of tracer in this location. They postulated:

Bruch's membrane may act as a constricting mechanical barrier to axoplasmic transport, especially if obstruction in deeper layers of the nerve head within the rigid scleral canal is present. In this regard, it is important that drusen of the disk are first observed clinically at the margins of the disk and histopathologically at the same location.

In studying mice retinas with hereditary degeneration of the visual receptor cells, a condition akin to human retinitis. pigmentosa, researchers26 found the rate of the fast component of axonal protein transport was normal; but the rate of slow transport was reduced by one third in the mutant mice, as compared with a closely. related strain of mice with normal retinas. The mutants had 20% fewer retinal ganglion cells and the remaining ganglion cells were reduced in size by 10 to 20%. We do not know if patients with retinitis pigmentosa have axoplasmic flow alteration, exhibit similar size changes, and show a decrease in number of their ganglion cells and axons. Although their retinal nerve fiber layer has been reported to. be normal,27 we have clinically observed nerve fiber laver defects.

Discussion

From our evidence we believe drusen of the disk form as a result of altered axoplasmic transport at the optic disk. Three factors favor this association: their location, their evolution, and their clinical and histopathologic similarity to processes in the optic disk known to chronically obstruct axoplasmic transport such as papilledema and enlarging melanocytoma.

Drusen form in the nerve fibers anterior to the lamina cribrosa. This is the site of the striking accumulation of intra-axonal material now recognized to occur in all forms of disk edema.²⁰ Furthermore, drusen initially develop at the disk edge where profound axonal swelling first occurs in disk edema.

In the familial neuropathy leading to drusen, we postulate that the normal physiologic damming of orthograde axoplasmic flow anterior to the lamina cri-

brosa²⁴ is aggravated by local factors. We suggest that one factor is the abnormally narrow aperture of the scleral canal. The accumulation of axoplasm as well as axonal swellings within this crowded space may account for the characteristic full, vellow-white appearance of the disk. The result may be nothing more than a structurally full disk composed of axons that are expanded, but not beyond the physiologic limits for long-term survival. Such structurally full disks are frequently found in members of families with drusen. But when this process exceeds these survival limits, degenerative changes begin which, in the course of many years, cause axonal death. By contrast, similar axonal atrophy occurs in the course of many months with chronic atrophic papilledema.

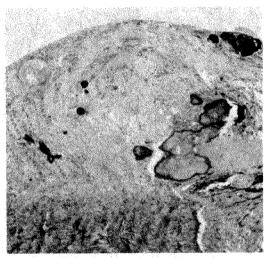
Seitz³ suggested that axonal interruption must precede calcification, which deposits in inspissated accumulations of axoplasmic material at the proximal edge of the interruption. Alternatively, stagnated material within uninterrupted axons may undergo calcification after it has passed a critical physical chemical state. Either supposition accounts for the absence of calcification early in life when few, if any, axons have been interrupted. Both explanations also support the welldocumented clinical observation that progressive calcium deposition occurs concurrently with diffuse retinal nerve fiber laver atrophy in later life.

We agree with Seitz that drusen of the disk are the end result of axonal deterioration. This proposal contradicts the commonly held concept that localized drusen form because of extra-axonal causes and then encroach upon axons to cause their degeneration.

For many years ophthalmologists have recognized anomalous branching and tortuosity of retinal vessels in patients with drusen of the optic nerve head and in such patients' family members.^{8,9} We pre-

sume these relate primarily to developmental processes associated with the small scleral canal and disk, and that additional modifications of the vascular pattern may occur as the drusen accumulate and enlarge. We have found no evidence to support the recently proposed hypothesis²⁸ that transudative vasculopathy is the primary cause of drusen.

By contrast, in eyes with retinitis pigmentosa, the optic nerve head characteristically appears waxy yellow and flat, and



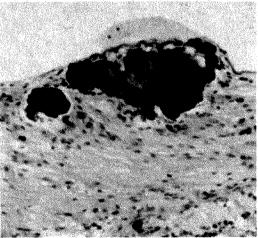


Fig. 9 (Spencer). Drusen of the disk in retinitis pigmentosa. Calcified structures are located in the prelaminar disk tissue and in the superficial parapapillary nerve fiber layer (left, hematoxylin and eosin, ×2.5; right, ×25) (From Cogan, D. G.²⁷).

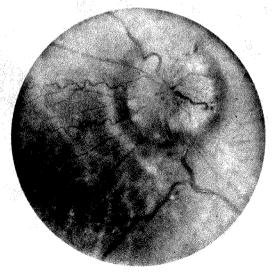


Fig. 10 (Spencer). Chronic atrophic papilledema. Focal accumulations of glistening drusen-like bodies lie in the superficial aspect of the swollen disk and partially obscure underlying vessels.

often lacks visible cupping. In comparison to eyes with familial drusen of the disk, the optic nerve head is not unusually small and vascular anomalies are uncommon. The waxy yellow ophthalmoscopic appearance of the disk and con-

cealed lamina cribrosa may be caused by stagnation of axoplasmic flow, which is initiated at the cell body rather than in the lamina cribrosa. The stagnation may result from reduced neural input from second order neurons. When drusen develop in patients with retinitis pigmentosa, they may also form in the superficial peripapillary nerve fiber layer, obscuring underlying vessels (Fig. 9). Their evolution is perplexing; once formed in this location they do not seem to change in size.

Papilledema causes chronic blockage of axoplasmic transport at the optic nerve head. In chronic atrophic papilledema, the disk appears yellow-white and focal accumulations of drusen-like bodies may become visible (Fig. 10). Their similarity to drusen may lead to diagnostic error. 29 However, in chronic atrophic papilledema, the yellow-white structures may represent aggregates of focal axonal swelling that can disappear when either the intracranial pressure is relieved or the atrophy advances (Fig. 11). Calcification does not occur, possibly because the process does not persist long enough.



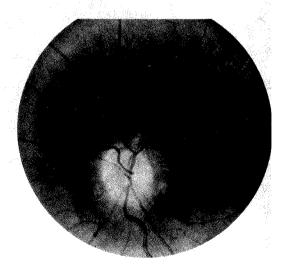


Fig. 11 (Spencer). Chronic atrophic papilledema. Preoperative (left) and postoperative (right) photographs of disk show disappearance of drusen-like bodies after relief of elevated intracranial pressure. The bodies were initially thought to represent drusen (From Okun, E.²⁹).



Melanocytomas of the optic nerve head, particularly those which are enlarging, may compress adjacent optic nerve head axons and produce a yellow-white elevation similar to that in drusen of the disk and papilledema (Fig. 12). Visual field loss may occur. Histologic examination of the disk in eyes enucleated because the tumor is considered a malignant melanoma reveals collections of swollen axons

adjacent to the tumor that we believe to be the anatomic substrate of the elevation (Fig. 13). To our knowledge, calcification has not been seen in a case of melanocytoma of the nerve head, possibly because axonal interruption is a rare event; however, vascular abnormalities and peripapillary, as well as disk, hemorrhages have been observed. In each condition—drusen, chronic papilledema, and enlarging

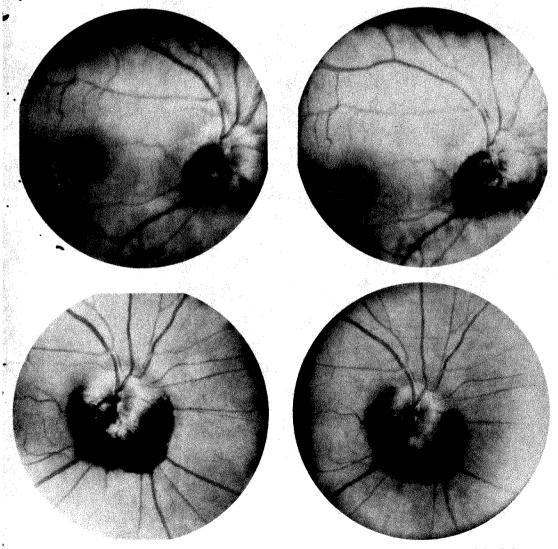
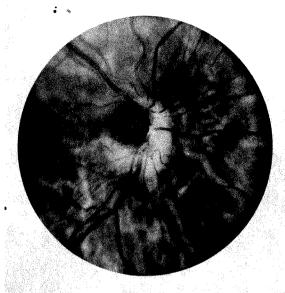


Fig. 12 (Spencer). Melanocytoma of optic nerve head. The enlarging tumor has compressed the disk tissue and produced a yellow white elevation. Top left, 1967; top right, 1968; bottom left, 1973; and bottom right, 1976 (From Cleasby, G.).



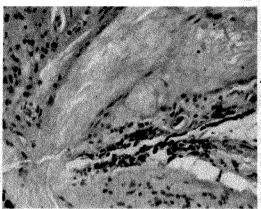
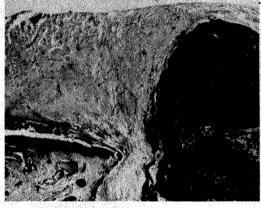


Fig. 13 (Spencer). Melanocytoma of optic nerve head. Top left, Case 1. Elevated disk tissue adjacent to tumor. Patient had marked visual field loss. Top right, Swollen axons in area corresponding to yellow white elevation (hematoxylin and eosin, ×40). Bottom right, Case 2. Swollen disk adjacent to melanocytoma. Patient had visual field loss and parapapillary hemorrhage (hematoxylin and eosin, ×2.5) (From Bec, P., European Pathology Society Meeting, 1973).



melanocytoma—alteration in the appearance and pattern of flow occurs through vessels of the optic nerve head.

SUMMARY

We believe axoplasmic transport alteration is the anatomic substrate for formation of drusen of the optic disk. In familial cases the cause of axoplasmic transport alteration may be related to the presence of a genetically determined, small, crowded optic nerve head. We believe these congenitally elevated nerve heads evolve over a period of many years through stages of atrophy and drusen formation. Vascular malformations in the

familial cases are primarily developmental; however, secondary vascular alterations may occur as the drusen enlarge. In retinitis pigmentosa the drusen may be caused by diminished production of axoplasmic material by the ganglion cell. Chronic alterations in axonal transport from any cause produce aggregates of swollen nerve fibers. These give a yellow-white appearance to the disk tissue and account for the yellow, filled-in appearance of the disk in patients with drusen, chronic atrophic papilledema, melanocytomas, and, in part, for the waxy yellow appearance of the disk in retinitis pigmentosa.

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RESULTS OF INTRAOCULAR LENS IMPLANT SURGERY

THE THIRD BINKHORST MEDAL LECTURE

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Appropriately, this lecture honors the man who revived intraocular lens implant surgery 20 years ago, Cornelius Binkhorst. It was once said of him: "His skill is only exceeded by his integrity." Scientific integrity remains the most urgent need to insure an orderly, sequential growth of intraocular lens implant surgery.

Intraocular implant lens surgery has generated controversy. This controversy persists partly because of insufficient data documenting advantages and risks of the procedure. This report summarizes the results of ten years' experience with more than 2,000 intraocular lens implantations. Although the surgery and implants used in this series may differ widely from those used by others, the painstaking dedication to data collection should provide beneficial information on the safety and efficacy of lens implantation for both implant and nonimplant surgeons.

MATERIAL AND METHODS

My major experience with intraocular lens implant surgery has involved the Copeland iris plane implant and the Binkhorst implants. The material in this study included the first 81 Copeland implants inserted between February 1968 and October 1969. After a two-year moratorium on lens implantation, I evaluated the next consecutive 81 Copeland im-

plantations, starting in October 1971. Thus, I subdivided 162 consecutive Copeland lens implantations into two series of 81 each. During the two-year moratorium, the Miami Community Protocol imposed restrictions on Miami lens implant surgeons, particularly regarding minimum age for the surgery, time interval between surgery on two eyes on the same patient, and provisions for exceptions. The indications for surgery were consequently more conservative in the later 81-case series.

I also studied the first 650 Binkhorst implants associated with intracapsular and extracapsular cataract extraction. The last patient in this series underwent surgery on April 8, 1977.

RESULTS

Copeland implants—With the collaboration of Lee Duffner, M.D., I performed an analysis of the first 81 Copeland lenses (Series 1) implanted before the 1969 moratorium and the next consecutive 81 implantations (Series 2) after the moratorium was lifted in 1971. Duffner performed all examinations. The results were published² and later updated.³ Series 1 had an average follow-up period of slightly more than six years, excluding those patients dead or lost to follow up (Table 1). The corresponding follow-up period for Series 2 was slightly less than three years. The patients in the later series were older (average age, 76.3 years) than those in the earlier series (average age, 70.3 years).

Visual acuity of 6/12 (20/40) or better was achieved in 74.1% of the eyes in Series 1 and 79.0% in Series 2, including those eyes from which the implant

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TABLE 1
PATIENT POPULATION*

•	Se	ries 1	Sei	ries 2
•	No. of Patients	Follow-up (mos)	No. of Patients	Follow-u _] (mos)
Dead	18	32	7	13
Lost to follow up	2 .	48	4	12
Available	61	· 75	7 3	32
Follow-up range		7-86		1-45
Mean age (yrs.)	70.3		76.3	

^{*}Each series comprised 81 patients.

was removed (Table 2). Implants were removed from ten of 162 eyes (6.2%) in these two series. Significantly, this occurred more frequently in the earlier series :(eight of 81; 9.9%) than in the later series (two of 81; 2.5%). Six of these ten eyes achieved visual acuity of 6/12 (20/40) or better by using an aphakic spectacle or à contact lens. Senile macular choroidal degeneration associated with an advanced cataract accounted for failure to achieve visual acuity of 6/12 (20/40) in 15 of 162 eyes (9.3%) (Table 3). Cystoid macular edema was responsible in 12 of 162 eyes (7.4%). This diagnosis was made by ophthalmoscopy and biomicroscopy without fluorescein angiography. The remaining causes occurred less frequently.

Copeland implants were placed in 13 eyes of 12 patients in Series 1 who were less than 60 years of age. By the time Series 2 began, more rigid criteria for case selection were in force in Miami. There-

fore, no patients in Series 2 were less than 60 years of age.

Five (38%) of the total Copeland im plants were removed because of uveitis. The time of implant removal for each cas respectively was four weeks, 13 months 18 months, 36 months, and 48 month after operation.

Of the eight eyes of patients under 6 years of age with the implant in situ, si (75%) had cystoid macular edema, al with visual acuity of less than 6/12 (20 40)

Four of the five patients who had a implant removed had a routine intracap sular extraction in the fellow eye. All eye had visual acuity of 6/7.5 (20/25) or bet ter. Three of the six eyes with cystoic macular edema had a routine intracapsular cataract extraction in the fellow eye None developed cystoid macular edema

The reason for the unsatisfactory re sults in patients under 60 years of age i

TABLE 2
RESULTS OF VISUAL ACUITY

	Series 1		Ser	ies 2
•	Lens In	Lens Out	Lens In	Lens Ou
6/6(20/20)—6/12(20/40)	55	5	63	1
6/15(20/50)—6/60(20/200)	15	2	13	1
Less than 6/120(20/400)	3	1	3	0
6/12(20/40) or better	74.	1%	79	0.0%

TABLE 3 Causes of visual acuity less than 6/12(20/40)*

	Series 1		Series 2		; '	
	Lens In	Lens Out	Lens In	Lens Out		
Senile macular degeneration	11	0	4	0		
Cystoid macular edema or pucker	6	2	4	0		
Diabetic maculopathy	Ó	0	1	0		•
Ischemic optic neuropathy	. 0	1	2	1		
Corneal edema	1	.0	2	0		
I.O.L. membrane	. 0	o	. 2	0		
. Glaucoma	0	0	0	* O		
Unknown	: 0	0	1	0		

^{*}I.O.L. designates intraocular lens.

uncertain. Since this has not been reported with other types of iris-supported imimplant.

After performing nearly 800 Copeland implants, I was not satisfied with them. I was disturbed by the cells in the anterior chamber, fixed pupils, retrolental membranes, as well as the high incidence of clinically significant cystoid macular ede-

Binkhorst implants—I returned to using the Binkhorst implant in early 1974: its design had been improved since I started using it in 1967. The anterior loops of the iris clip implant were shortened and both pairs of loops were bent backward at an angle of 10 to 15 degrees. Both modifications lessened the risk of corneal edema. Additionally, the lens was implanted in a vertical position and a transiridectomy suture was used to reduce the incidence of dislocation. The iridocapsular lens was used for extracapsular surgery at first. This two-loop lens had platinum-iridium loops. Later the iris clip implant was used in both intracapsular and extracapsular cases.

I subjected the Binkhorst implant to an intensive clinical evaluation. A relatively short-term study of the first 650 Binkhorst implants was made. The last case had more than a six-month follow-up. There

were 650 implantations in 630 patients (20 bilateral). The patients ranged in ageplants, it may be peculiar to the Copeland from 56 to 94 years. The mean age was 73.96 years. Nearly 90% of the patients were between 65 and 84 years old (Table 4); 4.8% were less than 65 years and 5.7% were more than 84 years of age.

> The type of cataract extraction in this series was heavily weighted toward the intracapsular technique, which was used in 559 (86%) of the cases. The extracapsular technique was used in 91 (14%).

> Visual acuity of 6/12 (20/40) was achieved in 89.8% of the eyes in this series (Table 5). Only 1.4% had less than 6/120 (20/400). Since the age range of the

TABLE 4 PATIENT POPULATION ACCORDING TO AGE GROUPS

Age (yrs)	No.	Percent of Total Cases
55-59	5	0.8
60-64	26	4.0
65-69	125	19.2
70-74	196	30.2
75-79	174	26.8
80-84	87	13.4
85-89	31	4.8
90-94	6	0.9
Total	650	100.0
Less than 65		4.8
65-84		89.5
Over 84		5.7

TABLE 5
VISUAL ACUITY RESULTS

Range	No.	Percent
3/5(20/15)6/12(20/40)	584	89.8
3/15(20/50)6/24(20/80)	29	4.5
3/30(20/100)—6/120(20/400)	28	.4.3
Less than 6/120(20/400)	9 ·	1.4
Гotal	650	100.0

patients was from 56 to 94 years, it was mportant to divide them into five-year age groups (Table 6). As expected, in-

creasing age was associated with poorer visual acuity, especially in the most populous groups aged 60 to 89 years.

The most common causes of failure to achieve visual acuity of 6/12 (20/40) in implant surgery were senile macular choroidal degeneration, cystoid macular edema, and corneal edema. Senile macular choroidal degeneration was the cause in 4.5% of the 650 eyes, cystoid macular edema in 1.4%, and corneal edema in 0.9%. Other causes were less frequent (Table 7).

I compared the visual acuity results and

TABLE 6 VISUAL ACUITY OF 6/12(20/40) OR BETTER BY AGE GROUPS

Age (yrs) No. of Patients	Percent of Total Cases	•		
55-59 4 60-64 25	80.0 96.2		,	
65-69 117 70-74 182	93.6 92.9			
75-79 151 80-84 76 85-89 23	$86.8 \\ 87.4 \\ 74.2$	÷ in		
90-94 6 Total 584 of 650	100.0 89.8	<i>,</i> .	,	

TABLE 7

Causes of visual acuity of less than 6/12(20/40) in 66 of 650 cases

	No. Percent of 650 Percent of 66		
	Senile macular degeneration 29 4.5 43.9	•	
	Cystoid macular edema 9 1.4 13.6		
· .	Corneal edema 6 0.9 9.1		
	Vein thrombosis 4 0.6 6.1		
•	Retinal detachment 3 0.5 4.5		- 2
	Diabetic maculopathy 2 0.3 3.0		
	Secondary membrane 2 0.3 3.0		
	Macular hole 1 0.2 1.5		
,	Macular pucker 1 0.2 1.5		
• '	Ischemic optic neuropathy 2 0.3 3.0		
	Glaucomatous optic atrophy 1 0.2 1.5		
•	Optic atrophy 1 0.2 1.5	,	,
	Retinitis pigmentosa 1 0.2 1.5		
	Amblyopia 3 0.5 4.5	•	•
+ 1	Unknown 1 0.2 1.5		
•	Total 66 10.2 100.0	•	,

TABLE 8 Visual acuity of less than 6/12(20/40) after lens implantation

Causes	Copeland Lens (%)	Binkhorst Lens (%) :	
Senile macular degeneration	15/162 (9.3)	29/650 (4.5)	,
Cystoid macular edema	12/162 (7.4)	9/650 (1.4)	
Corneal edema	3/162 (1.9)	6/650 (0.9)	٠
Visual acuity of 6/12(20/40) or better	124/162 (76.5)	584/650 (89.8)	

the causes of failure to achieve visual acuity of 6/12 (20/40) or better in the 162-case Copeland series and the 650-case Binkhorst series (Table 8). Although the follow-up period was shorter for the Binkhorst series, the visual acuity results were much better; 89.8% achieved 6/12 (20/40) or better visual acuity, as com-

TABLE 9
BILATERAL IMPLANTS*

Bilateral Combination	No.
Copeland - Binkhorst ICCE	18
Copeland - Binkhorst ECCE	$\dot{2}$
Worst - Binkhorst ICCE	7
Fyodorov type 1 - Binkhorst ICCE	1
Binkhorst ICCE - Binkhorst ICCE	15
Binkhorst ICCE - Binkhorst ECCE	5
Binkhorst ECCE - Binkhorst ECCE	2
Total	50

^{*}The patient age range was 66 to 91 years; the mean age was 77.3.

TABLE 10
REASONS FOR IRIDOPLASTY IN 25 (3.8%) OF 650 CASES

	No. of Cases
Miotic therapy	11
Previous peripheral iridectomy	11
Previous sector iridectomy	2
Previous iridencleises	1 .

pared to 76.5% in the Copeland series. The incidence of clinically significant cystoid macular edema was much lower in the Binkhorst series (1.4%) than in the Copeland series (7.4%).

At the time of this report, 50 bilateral implants had been performed (Table 9), The mean age of these patients was 77.30 years, as compared to 73.96 years for the 650-case Binkhorst series. The most frequent bilateral combination was a Copeland implant in one eye and a Binkhorst implant in the other eye, both with an intracapsular cataract extraction.

The number of bilateral implants is small for such a large series. Although 650 Binkhorst implants were placed in 630 patients, (20 bilateral cases), these figures indicate that 22 patients had Binkhorst lenses in both eyes. This discrepancy exists because two patients had a Binkhorst implant placed in the first eye in 1967.

Some patients required an iridoplasty in association with the lens implantation. This was usually caused by a miotic pupil in eyes being treated for glaucoma or by a miotic pupil in eyes after a peripheral iridectomy. A total of 25 eyes (3.8%) required an iridoplasty (Table 10). Failure to achieve visual acuity of 6/12 (20/40) in two of these 25 cases was caused by senile macular choroidal degeneration.

Vitreous disturbance during surgery in this series occurred in 49 of the 650 eyes (7.5%). Any disruption of the anterior

[†]ICCE designates intracapsular cataract extraction; ECCE, extracapsular cataract extraction.

TABLE 11
OPERATIVE VITREOUS PROBLEMS
IN 49 OF 650 CASES*

	No.†	Percent
During ICCE During ECCE Before ICCE During implantation Through peripheral iridectomy Aspiration for bulge Total	9/559 9/91 1/559 17/559 8/559 5/559 49/650	1.6 9.9 0.2 3.0 1.4 0.9 7.5

^{*}ICCE designates intracapsular cataract extraction; ECCE, extracapsular cataract extraction.

surface of the vitreous, accidental or intentional, was included as a vitreous disturbance (Table 11). The incidence of vitreous loss during the intracapsular cataract extraction was 1.6%. It was 9.9% with extracapsular extraction. The high figure for the latter is attributed to early experience with the newer automated techniques of extracapsular extraction. Vitreous disturbance occurred most frequently during the implantation of the pseudophakos. In some instances the vitreous face was ruptured through the peripheral iridectomy during the placement of the transiridectomy suture. Early in the series, aspiration of retrovitreal aqueous was performed in some instances because - of vitreous bulge immediately after the cataract was extracted. Of these 49 eyes, 77.6% achieved visual acuity of 6/12 (20/ 40) or better, as compared to 89.8% in the overall series (Table 12).

Most pioneer lens implant surgeons recommend avoiding lens implantation when operative loss of vitreous occurs. I disagree with this view, provided the surgeon is capable of managing vitreous loss according to modern techniques. Once operative loss of vitreous has occurred, lens implantation does not alter the inci-

TABLE 12

CAUSES OF FAILURE TO ACHIEVE VISUAL ACUITY OF 6/12/(20/40) IN 11 OF 49 EYES WITH OPERATIVE VITREOUS PROBLEMS

Cause	No.
Senile macular degeneration	4
Cystoid macular edema	. 2
Retinal detachment	1
Corneal edema	1
Diabetic maculopathy	1
Amblyopia	1
Unknown.	1
Total	(22.4%) 11

dence of postoperative cystoid macular edema and retinal detachment. The incidence of both of these complications increases whether or not lens implantation is performed.

In this series, there were seven instances of clinically significant cystoid macular edema (visual acuity of less than 6/12 (20/40)) in the 601 eyes (1.2%) without an operative disturbance of vitreous. Two (4.7%) of the 49 eyes with an operative vitreous disturbance had cystoid macular edema.

Additional surgery was required in 44 of the 650 eyes (6.8%). This was mainly caused by the development of a secondary membrane (Table 13).

Secondary membranes occurred in both extracapsular and intracapsular cases and were dense enough to require surgery in 26 of the 650 eyes (4.0%). This number will likely increase with increasing post-operative time. The most frequent operation for secondary membrane was a discission. In more severe cases where the iris was drawn in to create a pincushion pupil and where the anterior vitreous was opacified, a trans pars plana membranectomy and vitrectomy were performed. Of 91 extracapsular cases, surgery was necessary in 15 or 16.5% (Table 14). This included 14 discissions and one trans

[†]There were 559 ICCEs and 91 ECCEs.

TABLE 13
Additional surgery in 44 (6.8%)
OF 650 cases*

Procedure	No.
Discission secondary membrane	20
TPPV for secondary membrane	6
Penetrating keratoplasty	5
Retinal detachment	5
Wound repair	3
Implant removal	2
Implant replaced	1
Malinsertion corrected	· Ì

^{*}TPPV designates trans pars plana vitrectomy.

pars plana vitrectomy. Of the 559 intracapsular cases, surgery was necessary in 11 (2.0%). This included six discissions and five trans pars plana vitrectomies.

All 14 extracapsular cases requiring a discission and the one requiring trans pars plana membranectomy and vitrectomy achieved visual acuity of 6/12 (20/40) or better. The six intracapsular cases that required a discission also achieved visual acuity of 6/12 (20/40) or better. Three of the five intracapsular cases requiring a trans pars plana vitrectomy failed to achieve visual acuity of 6/12 (20/40), two because of cystoid macular edema and one because of ischemic optic neuropathy.

Dislocations of the implant occurred in 16 cases (2.5%). In most instances the implant was easily repositioned by using

a needle placed in the anterior chamber through a corneoscleral limbal perforation. In two instances, the implant was repositioned pharmacologically. In two cases, no treatment was necessary. In one, an iridocapsular implant dislocated completely into the anterior chamber. Because the posterior capsule was intact, the implant was removed and an iris clip implant was safely inserted. In no instance did an implant dislocate into the vitreous cavity.

Retinal detachments occurred in four of the 650 cases (0.62%). Henry Clayman, M.D., examined our office records of 1,651 consecutive intracapsular cataract extractions between 1968 and 1970. Daniel Eichenbaum, M.D., examined the office records of 653 consecutive intracapsular cataract extractions between 1973 and 1974. We compared the total 2,304 routine cataract extractions and the 650. Binkhorst implants with regard to retinal detachment (Table 15).

From these data one may conclude that the age of patients who develop a retinal detachment after a routine intracapsular cataract extraction (62.23 years) of after a lens implantation (67.75 years) is lower than the mean age of the patients in each group. The rate of retinal detachment after routine intracapsular cataract extraction (1.74%) is nearly three times as great as after lens implantation (0.62%); but this probably results from case selection for each procedure. Eighteen of the 40.

TABLE 14
Surgery for secondary membranes*

,	No. of Cases		Discission	TPPV	Total (%)
ECCE	91		14	1	15 (16.5)
ICCE	559	٠.	6	5	11 (2.0)
ECCE + ICCE	650		20	6	26 (4.0)

^{*}TPPV designates trans pars plana vitrectomy; ECCE, extracapsular cataract extraction; ICCE, intracapsular cataract extraction.

TABLE 15
RETINAL DETACHMENTS*

`		ICCE	IOL	
	Cases	2,304	650	
•	Retinal detachments	40 (1.74%)	4 (0.62%)	
•	Mean age of retinal detachment patients	62.23	67.75	
	Myopia with aphakic refraction	18/40		
	+9.00 diopters or less			
	Time of occurrence of retinal detachment (postop.)	•		
•	0-6 mos	27/40	1/4	
	6-12 mos	4/40	1/4	
	Greater than 1 yr	9/40	2/4	

^{*}ICCE designates intracapsular cataract extraction; IOL, intraocular lens surgery.

retinal detachments in the former group occurred in patients who were myopic (aphakic refractive error of +9.00 diopters or less). Many young patients were also in this group. In both instances, these patients would not have been accepted by me for lens implantation.

I have also studied the problem of dislocations and whether the transiridectomy suture is really necessary.

Worst⁴ was the first to utilize a suture to prevent dislocation of a Binkhorst iris slip implant. He sutured the anterior superior loop to the iris. One year later, Binkhorst⁵ introduced a transiridectomy suture for the same purpose.

Through my experience with the Binkhorst implant in over 800 cases, I have concluded the placement of the transiri--dectomy suture is one of the most difficult technical aspects of the surgery. It cannot be placed in a closed eve under air. It requires direct visualization to achieve proper insertion and the desired space between the anterior and posterior loops. The placement of the suture often ruptures the vitreous face through the iridectomy. Because the cornea must be retracted to some degree, some contact between the optical portion of the implant and the corneal endothelium may occur. Beauchamp, Clayman, and Jaffe⁶ and Alpar⁷ suggested variations in the technique that simplified the procedure, but did not make it sufficiently safe.

At the suggestion of Miles Galin, M.D., (personal communication), I eliminated the suture in my Binkhorst implant technique. Galin recommends a careful, controlled postoperative dilation of the pupil to lessen the incidence of synechiae of the iris to the posterior loops of the implant. I do not dilate the pupil postoperatively (Table 16).

I hesitate to draw absolute conclusions

TABLE 16
DISLOCATIONS WITH AND WITHOUT
TRANSIRIDECTOMY SUTURE*

Type of	No. of	-	
Extraction	Cases	Dislocations	Percent
. Iri	idocapsular	(2-loop) Implant	***************************************
ECCE	33	3	9.1
Transirio	lectomy Su	ture - Iris Clip In	plant
ECCE	52	0	0.0
ICCE	413	9	2.2
Total	465	. 9	1.9
. N	o Suture - I	ris Clip Implant	
ECCE	`7	0	0.0
ICCE	235	5	2.1
Total	242	5	2.1
Coml	oined Series	- Iris Clip Impla	int
ECCE .	59	0	0.0
ICCE ·	648	14	2.2
Total	707	14	2.0

^{*}ECCE designates extracapsular cataract extraction; ICCE, intracapsular cataract extraction.

from these results because this is a shortterm series. The follow-up period for the 242 cases without a transiridectomy suture is shorter than the 465 cases with a suture. In my experience, however, dislocations usually occur early in the postoperative period. Only one dislocation in the suture series occurred more than six months after the surgery. Tentatively, I conclude that the use of the transiridectomy suture does not reduce the incidence of postoperative dislocation of the Binkhorst iris clip implant. The transiridectomy suture may favor dense adhesions of the iris sphincter to the two superior loops of the implant. This tends to make dislocation of the inferior loops more likely. Two advantages of the transiridectomy suture are that it prevents both rotation of the implant and dislocation of the entire implant into the vitreous. In three cases of the suture-less series, the iris clip implant rotated to the horizontal position within the first postoperative week.

The current model of the Binkhorst iris clip implant with its 10- to 15-degree posterior loop orientation may tend to make dislocation less of a danger than the older Binkhorst implant with the same loop lengths. Although originally intended to lessen the risk of corneal edema, the new model may have the additional advantage of providing more secure fixation with the eye.

In this personal series involving three instances of dislocation of an iridocapsular (two-loop) implant in 33 cases and 13 dislocations in 707 iris clip (four-loop) implants, there was only one complete dislocation into the vitreous (in a nonsutured implant) and not a single case of corneal edema resulted from implant dislocation. Two patients had implants that dislocated twice; no other patient had more than one dislocation. It was not necessary to use a McCannel suture⁸ in any case.

The follow-up period in this series was

relatively short, especially for the sutureless cases. Therefore, one may not yet absolutely conclude that the transiridectomy suture is obsolete.

I have been engaged in a cystoid macular edema study since May 1974. The study compares the incidence of cystoid macular edema, as determined by fluorescein angiography, in patients 67 years of age or older who have undergone the following: (1) intracapsular cataract extractions with the implantation of a Binkhorst iris clip lens; (2) extracapsular cataract extraction with the implantation of a Binkhorst iridocapsular or iris clip lens; or (3) intracapsular cataract extraction without an implant.

All surgery was performed by one surgical group and the angiograms were read by retinal subspecialists. The angiogram readers had no knowledge of the kind of surgery performed and the surgeons did not see the angiograms. Fundus angiograms were taken at four, eight, and 16 to 24 months postoperatively.

The study included 190 Binkhorst implants; 145 intracapsular and 45 extracapsular cases. Controls were 113 routine intracapsular extractions without an implant. Some patients did not complete the study because of inadequate studies, death or severe illness, intravenous fluorescein reactions, onset of other fundus disease (senile macular choroidal degeneration, diabetic maculopathy, retinal detachment, retinal branch vein thrombosis, and the like), lack of reviewer agreement in questionable cases, poor quality angiograms, and noncompliance.

The results indicate that routine intracapsular cataract extractions and all implants have a comparable incidence of cystoid macular edema at four, eight, and 16 to 24 months postoperatively (Table 17).

Extracapsular Binkhorst implants (all with an intact posterior capsule) appear to have a significantly lower incidence of

TABLE 17
INCIDENCE OF CYSTOID MACULAR EDEMA*

	:		4 Mos (%)	8 Mos (%)	16-24 Mos (%)	 •
		All implants	27/190 (14.2)	16/158 (10.1)	18/138 (13.0)	1
	•	Controls				
\$2.5 €		ICCE implants				
•		ECCE implants	3/45 (6.7)	2/45 (4.4)	1/29 (3.4)	

^{*}ICCE designates intracapsular cataract extraction; ECCE, extracapsular cataract extraction.

cystoid macular edema than an intracapsular cataract extraction either with or without a Binkhorst implant (Table 17). A larger number of extracapsular implants may be necessary to establish clinical significance.

Less than 20% of eyes that had at least one positive fluorescein angiogram at any time during the study had less than 6/12 (20/40) visual acuity (Table 18). Although these results indicate good visual acuity in all groups with cystoid macular edema, the acuity did not take into account the problem of distortion.

This study, which is still in progress, will be reported by all the participants then it is completed.

DISCUSSION

I believe we must document what we have done and justify why we are doing it because facts can only arise from carefully collected data.

No one has satisfactorily presented a

TABLE 18
FINAL VISUAL ACUITY OF EYES WITH
CYSTOID MACULAR EDEMA AT ANY
TIME DURING STUDY

	No. of Patients With Positive Study	No. With V.A. Less Than 6/12(20/40) (%)
All implants	34	6 (17.7)
Controls	26	5 (19.2)

^{*}V.A. designates visual acuity.

long-term follow up on a particular implant used with a particular technique. Implant surgeons who presently favor an iris-supported implant justify its safety by stating that Binkhorst's iris clip implant has been successful for 20 years.

The early iris clip implant with its long 9- to 9.5-mm anterior loops placed horizontally frequently caused late corneal edema. Shortening the loops increased the risk of implant dislocation. The implant was placed in the vertical position. A transiridectomy suture was suggested. Finally, the implant design was changed so that the anterior and posterior loops were curved posteriorly at an angle of 10 to 15 degrees. This latest model has only been with us for about six years. This does not outline the entire Binkhorst story. Because of fixation problems and the threat of corneal edema, Binkhorst turned to capsular fixation. The justification for this was later changed by the assumption that the posterior capsule was responsible for preventing cystoid macular edema. The loops of the iris clip implant, originally made of nylon, are now being made of polypropylene; its advantages have not been clinically substantiated. The loops of the iridocapsular implant, at first nylon, later platinum-iridium, then titanium, and now polypropylene have also been frequently modified.

The Choyce Mark VIII implant has recently captured the fancy of many implant surgeons. Proponents of the Mark VIII implant proudly assert that this latest version has remained unchanged since 1963. However, how many surgeons, aside from the originator, have used it for more than two years? No long-term retrospective study of this implant has been done that would satisfy the demands of epidemiologists and biostatisticians. Tragic instances of inadequate quality control of this implant have occurred in the United States. This is certainly not the fault of Choyce. The Mark VIII implant is relatively easy to insert. However, even the most ardent enthusiast would admit that its insertion into the anterior chamber angle is a blind procedure. Roper-Hall⁹ showed the family tree of implants for the first time at the 1975 American Academy of Ophthalmology and Otolaryngology meeting. Normally a tribute to progeny, this history is a documentation of our past failures.

I do not wish to criticize the pioneers of implant surgery. Without Binkhorst there would be no lens implant surgery; and Choyce has apparently succeeded where others failed. All progress is marked by a continuum of changes. I wish only to remind my colleagues to keep the intraocular implant lens in perspective, to exercise mature judgment, and not to follow new concepts indiscriminately. The intraocular implant lens is not for every surgeon or every patient, but it has been the most exciting advance in cataract surgery in our lifetime.

SUMMARY

I compared an early series of 162 consecutive Copeland intraocular lens implants with a later series of 650 consecutive Binkhorst implants. Of the eyes with Copeland implants, 76.5% achieved 6/12 (20/40) or better visual acuity compared to 89.8% in the Binkhorst series. There

was a striking difference in the incidence of clinically significant cystoid macular edema (less than 6/12 [20/40], visual acuity); 7.4% in the Copeland series and [1.4% in the Binkhorst series.

I studied the Binkhorst series for visual acuity, causes of less than 6/12 (20/40) visual acuity, operative vitreous problems, additional surgery, secondary membranes, dislocations, and retinal detachment

A fluorescein angiographic study of 190 Binkhorst implants and 113 aphakic controls was done. There was a comparable incidence of cystoid macular edema in both groups at four, eight, and 16 to 24 months postoperatively. Extracapsular Binkhorst implants showed a lower incidence than intracapsular Binkhorst implants.

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A COMPARISON OF 500 BINKHORST IMPLANTS WITH 500 ROUTINE INTRACAPSULAR CATARACT EXTRACTIONS

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Little data have been published comparing the results of intraocular implant lens surgery (IOL) and routine intracapsular cataract extraction (ICCE). Therefore, we completed a retrospective study of these two groups by first matching them as closely as possible.

MATERIAL AND METHODS

The protocol was as follows:

- 1. All surgery was performed by the same surgical group.
- 2. The surgery was performed during a similar time span.
- 3. There were 512 consecutive Binkhorst implants with 12 omissions.
- 4. There were 653 consecutive intracapsular cataract extractions with 153 omissions.

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5. This left a net of 500 in each group for comparison.

Since we studied consecutive cases, it was necessary to omit cases from each group to age match them and to eliminate cases from the ICCE group that would not have qualified for the IOL group by our standards. We determined several reasons for omission (Table 1).

Patients with either moderate to high myopia, glaucomatous optic atrophy, previous retinal detachment, or only one eye were not considered qualified for an intraocular lens. Combined surgery in the ICCE group referred to combined ICCE and trabeculectomy or combined ICCE and penetrating keratoplasty. In the IOL group, this referred to combined IOL and penetrating keratoplasty. The study involved patients over the age of 60 years (Table 2).

The patients in both groups had their surgery during a nearly equal time span and at a time when we were performing

TABLE 1
Omissions*

	ICCE	IOL		,
Patients less than 60 years old	74	3	*** **********************************	
Myopia (aphakic refraction +6.00 D or less)	31	0		
Follow-up period too short	22	3		
Glaucomatous optic atrophy	11	1		
Previous repair of retinal detachment	3	0		
Combined surgery	7	5		
One-eyed patient	5	0		
Total	153	12		

^{*}ICCE designates intracapsular cataract extraction; IOL, intraocular lens implant surgery.

:

TABLE 2
PATIENT POPULATION IN EACH SURGICAL GROUP

	ICCE	IOL
No. of patients	500	500
Age range	60-90 yrs	60-90 yrs
Mean age	72.72 yrs	73.95 yrs
Time span	30 mos	31 mos

IOL surgery in about 44% of our cataract extractions.

RESULTS

Visual acuity results were similar in both groups (Table 3). We compared those patients in each surgical group who achieved 6/12 (20/40) or better according to five-year age groups (Table 4). Dividing the patients into five-year age groups revealed little more than we already knew when we considered all 500 cases in each surgical group as a whole. However, it did reveal a tendency to poorer final visual acuity with increasing age in both groups.

We studied four causes of failure to achieve visual acuity of 6/12 (20/40) or better in each group; senile macular degeneration was the most common cause of failure, accounting for 7.6% in the ICCE group and 4.2% in the IOL group (Table 5). Of the 73 patients in the ICCE group

TABLE 3 Visual acuity results after surgery

	No. ICCE (%)	No. IOL (%)
6/5(20/15)— 6/12(20/40)	427 (85.4)	445 (89.0)
6/15(20/50)— 6/24(20/80)	42 (8.4)	22 (4.4)
6/30(20/100)— 6/120(20/400)	23 (4.6)	24 (4.8)
6/120(20/400)	8 (1.6)	9 (1.8)
Total	500 `	500

who did not achieve 6/12 (20/40) visual acuity, senile macular degeneration was the cause in 52%. Of the 55 patients in the IOL group who did not achieve 6/12 (20/40) visual acuity, senile macular degeneration was the cause in 38%.

The second most frequent cause in both groups was cystoid macular edema, accounting for 2.2% of failure in the ICCE group and 1.4% in the IOL group. Of those who failed to achieve 6/12 (20/40) visual acuity in both groups, cystoid macular edema was the cause in nearly equal incidence; 15% for the ICCE group and 13% for the IOL group.

There was a significant difference between the groups when corneal edema was considered. Although the numbers were small, the incidence of corneal edema as the cause of failure to achieve 6/12 (20/40) visual acuity was three times greater in the IOL group; 1.2% compared to 0.4% in the ICCE group. Corneal edema was the cause in 3% of the 73 cases. in the ICCE group and 11% of the 55 cases in the IOL group. This difference may be even greater than that indicated in this study since cornea guttata may have been a cause for performing a routine intracapsular cataract extraction rather than a lens implantation. However, the study did not take this factor into account.

The fourth cause of failure to achieve 6/12 (20/40) visual acuity was retinal detachment. The incidence was nearly-the same in each group; 0.8% of the 500 ICCE cases and 0.6% of the IOL cases. Of those who did not achieve 6/12 (20/40) visual acuity, retinal detachment was the cause in 5% in each group. When moderate to high myopic individuals and young patients were eliminated from the ICCE group, the rate of retinal detachment was about the same as for the IOL group, which was a preselected group (excluding young patients, moderate to high myopic patients, and the like).

The remaining causes appear unrelated

TABLE 4 VISUAL ACUITY RESULTS OF 6/5(20/15) TO 6/12(20/40) IN FIVE-YEAR AGE GROUPS

` .	•		ICCE		· · · · · · · · · · · · · · · · · · ·	· IOL			
-: 	Age Groups (yrs)	No. of Cases	6/5(20/15)- 6/12(20/40)	Percent	No. of Cases	6/5(20/15)- 6/12(20/40)	Percent		
, • 1 •	60-64	47	40	85.1	.25	24	96.0	·.	
,	65-69	106	99	93.4	95	87	91.6		
A	70-74	132	117	88.6	151	′ 139	92.1	*	-10
	75-79	132	117	88.6	134	117	87.3	٠.	*
	. 80-84	70 -	47	67.1	65	56	86.2		
	85-89	12	7	58.3	27	19	70.4	,	
	90	1	0	0	3	3	100.0	11	
	Total	500	427	85.4	500	445	89.0	• •	•

to the type of surgery performed. The numbers are small for each group.

The incidence of the four causes of failure to achieve 6/12 (20/40) visual acuity (senile macular degeneration, cystoid macular edema, corneal edema, and retinal detachment) were considered for successive five-year age groups (Table 6).

The influence of age on these four causes of failure to achieve 6/12 (20/40) visual

acuity told us what we expected. Senile macular choroidal degeneration increases with increasing age. All reginal detachments occurred in patients under 70 years of age. There was no direct relationship between cystoid macular edema or corneal edema and age.

Discussion

A careful evaluation of the results and

TABLE 5

Causes of visual acuity results less than 6/12(20/40)

	ICCE			IOL :	
	% of 500 No.	% of 73	% of 500	No. %	of 55
Serile macular degeneration Cytoid macular edema Comeal edema Refinal detachment Vein thrombosis Dizbetic maculopathy Vitteous hemorrhage Macular pucker Macular hole Retinitis pigmentosa Optic atrophy Glaucomatous atrophy Ischemic optic neuropathy Amplyopia Congenital nystagmus Secondary membrane Expulsive hemorrhage Unfinown	(7.6) 38 (2.2) 11 (0.4) 2 (0.8) 4 0 3 3 2 1 0 1 0 0 1 2 1	(52) (15) (3) (5)'	(4.2) (1.4) (1.2) (0.6)	21 7 6 3 4 1 0 1 1 1 1 2 3 0 2	(38) (13) (11) (5)

TABLE 6

Causes of failure to achieve visual acuity of 6/12(20/40) according to age groups*

		ICCE		•
Age (yrs)	No. of SMCD (%)	No. of CME (%)	No. of CE (%)	
60-64 65-69 70-74 75-79 80-84 85-89 90	0 (0) 2 (1.9) 6 (4.5) 8 (6.1) 19 (27) 2 (17) 1 (100)	4 (8.5) 1 (0.9) 2 (1.5) 2 (1.5) 0 (0) 2 (17) 0 (0)	0 (0) 0 (0) 2 (1.5) 0 (0) 0 (0) 0 (0) 0 (0)	3 (6.4) 1 (0.9) 0 (0) 0 (0) 0 (0) 0 (0) 0 (0)
	•	IOL	,	
60-64 65-69 70-74 75-79 80-84 85-89 90	0 (0) 3 (3.2) 2 (1.3) 5 (3.7) 6 (9.2) 5 (19) 0 (0)	1 (4.0) 0 (0) 1 (0.7) 4 (3.0) 1 (1.5) 0 (0) 0 (0)	0 (0) 1 (1.1) 2 (1.3) 3 (2.2) 0 (0) 0 (0) 0 (0)	0 (0) 3 (3.2) 0 (0) 0 (0) 0 (0) 0 (0) 0 (0)

^{*}SMCD designates senile macular degeneration; CME, cystoid macular edema; CE, corneal edema; RD, retinal detachment.

complications associated with intraocular implant lens surgery is useful because there are few reports of this information. Our study was more meaningful because the results were compared with a standard method of cataract extraction without an implant. To be useful, such a retrospective study should match the two groups as closely as possible regarding surgeons, surgical technique, age of patients, time of surgery, and ocular status; the cases should also be consecutive. Our study will be further updated by comparing the visual acuity results and complications at specific time intervals.

SUMMARY

We compared 500 Binkhorst lens implants with 500 routine intracapsular cataract extractions. The two groups consisted of consecutive cases matched according to surgeons, surgical technique, time interval of the study, and ocular status.

The results of visual acuity were comparable in the two groups; 89.0% of the implant group and 85.4% of the routine intracapsular group achieved 6/12 (20/40) or better visual acuity.

The rate of complications in each group was comparable except in the case of corneal edema, which was higher in the implant group (1.2%) than in the routine intracapsular group (0.4%). The rate of clinically significant cystoid macular edema was higher in the routine intracapsular group (2.2%) than in the implant group (1.4%).

PREVALENCE OF SENILE CATARACT, DIABETIC RETINOPATHY, SENILE MACULAR DEGENERATION, AND OPEN-ANGLE GLAUCOMA IN THE FRAMINGHAM EYE STUDY

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According to the data from the Model Reporting Area, senile cataract, chronic simple glaucoma, senile macular degeneration, and diabetic retinopathy are the major causes of adult blindness in the United States today. Few surveys have been designed to study these diseases in the general population. Instead, studies tend to concentrate on eye clinic patients. The representative group of volunteers who have participated in the Framingham Heart Study² since 1949 gave us a unique opportunity to determine the prevalence of these major causes of blindness in an elderly population. In implementing this study, we emphasized standardized observations by trained examiners following a detailed protocol.

SUBJECTS AND METHODS

Of the 3,977 surviving members of the original Framingham Heart Study population, 2,675 members, ranging in age from 52 to 85 years in 1973, volunteered

for participation in the Framingham Eye Study. Detailed descriptions of this study have been published.^{3,4} The ocular examination consisted of two parts. Initially, a preliminary screening examination was performed. This included a short ophthalmic history, determination of best corrected visual acuity, applanation tonometry, and after mydriasis, examination of the lens by slit-lamp biomicroscopy and the macula by direct ophthalmoscopy, as well as measurement of the cup: disk ratio by indirect ophthalmoscopy. Gonioscopic examination was performed if narrow angles were suspected. The second part consisted of a definitive examination, by a senior observer, of those patients with evidence of any of the four diseases under consideration.

If the patient was aphakic or had lens changes characteristic of senile cataract (posterior subcapsular or cortical changes or nuclear sclerosis) in association with a best corrected visual acuity of 6/9 (20/30) or worse, we referred him to a more experienced definitive examiner for an extensive corroborative cataract evaluation. Prevalence data for senile cataract were based on the judgement of the definitive examiner. We used historical data and the results of the ocular examination to exclude patients with congenital cataracts or secondary cataracts caused by trauma, uveitis, or the like.

A patient was declared a glaucoma sus-

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pect on the basis of the screening examination if any of the following were noted in either eye: (1) history of previous diagnosis or treatment for glaucoma; (2) anterior chamber angle closed or less than 10 degrees in any quadrant; (3) average intraocular pressure (IOP) of 22 mm Hg or greater, recorded by applanation tonometry; (4) average pressure difference of 3 mm Hg or greater between the two eyes and IOP of 16 mm or more in one eye; (5) cup:disk ratio of 0.5 or greater in the horizontal or vertical meridian; or (6) the cup: disk ratio in the two eyes differed by 0.2 or more. Glaucoma suspects were referred for a definitive examination, including repeat applanation tonometry, gonioscopy, and visual field testing for glaucoma screening by a modification of Armaly's technique. 5 Perimetry was used to detect the characteristic visual field defects associated with open-angle glaucoma (such as blind spot enlargement of the Seidel type, arcuate scotoma, paracentral scotoma, nasal step, or advanced glaucomatous defect). We defined openangle glaucoma as the presence of any of the above field defects in those glaucoma suspects without angle abnormalities, lens dislocation, intraocular tumor, congenital disorders, uveitis, or other conditions precluding a diagnosis of angle-closure, secondary or congenital glaucoma.

Patients were referred for a definitive examination if the screening examiner, using direct ophthalmoscopy, identified or suspected the presence of microaneurysms or dot hemorrhages. We considered individuals positive for diabetic retinopathy if the definitive examiner reported they had any of the following: (1) retinal hemorrhages or microaneurysms; (2) soft or hard exudates; (3) intraretinal microvascular abnormalities; (4) macular edema; or (5) neovascularization of characteristic number and distribution, with noncontributory history of other predisposing conditions (such as systemic hypertension, blood dyscrasias, hyperviscosity states). The evaluation of changes consistent with diabetic retinopathy was based on a modification of the protocol used in the Collaborative Diabetic Retinopathy Therapy Trials. Each characteristic abnormality was illustrated in standard fundus photographs available as a reference to the examiner.

Similarly, the screening examiner referred for a definitive examination all patients with definite or suspected drusen or macular pigment disturbance when the best corrected visual acuity in the affected eye was 6/9 (20/30) or worse. Persons were also referred for definitive examinations if they had definite or suspected elevation of the pigment epithelium or the neurosensory retina, or perimacular circinate exudates. The definitive examination included inquiry and examination for evidence of uveitis, intraocular surgery or tumors, ocular trauma, hypotony, retinal vein occlusion, diabetes mellitus, sun gazing or retinal detachment, and factors that might cause macular pigmentary disturbance. The diagnosis of senile macular degeneration was made if the definitive examiner observed degenerative changes of either the dry type (includes pigment disturbance or drusen formation) or the exudative type (includes elevation of the pigment epithelium or neurosensory retina), and if the results of examination were noncontributory for a congenital, secondary, or other cause. Thus, we made the diagnosis of each disease on the basis of exclusion.

It was not possible to achieve 100% success in examining the total population, nor was it possible to obtain the continued participation of all subjects selected for the definitive examination. True prevalence rates can rarely be determined and our data are no exception. Therefore, we initially calculated prevalence rates under three alternative assumptions,^{3,4} providing a range within which the true value probably lies:

1. All persons not screened and all sus-

pects with incomplete status (either failed to take the definitive examination or to return for a glaucoma recall examination) have no disease.

- 2. All persons for whom final diagnosis was not completed are negative for disease, and those not screened have the same disease prevalence as those who were.
- 3. Suspects for whom a final diagnosis. was not completed have the same disease prevalence as suspects with final diagnoses, and those not screened are similar in disease prev-. alence to those who were.

Method 1 almost certainly provides a prevalence estimate that is too low. Most studies show that nonparticipants tend to have higher disease rates than participants. Method 3 assigns the same disease rates to the nonparticipants and those who did not have a definitive examination as were found in the examined group. While this probably still underestimates the true prevalence, of the three methods, it most likely approximates the true value. Further discussion of prevalence will present data calculated under the assumptions outlined in method 3.

Approximately one fourth of the surviving Framingham Heart Study cohort had moved outside the Framingham area, defined by a radius of approximately 25 miles from the city (Table 1). We examined 84% of those living within the immediate Framingham environs, but only 19% of those living outside the area. Data are presented for the former group only.

The association between senile cataracts and age is demonstrated (Table 2). Our data show a prevalence of 4.6% at age 52 to 64 years, 18% at 65 to 74 years, and 46% at age 75 to 85 years. In each age group, women have higher prevalence within reasonable limits of chance fluctu-

TABLE 1 FRAMINGHAM EYE STUDY POPULATION

Residence	Age (yrs)	No. Eligible	No. Examined (%)
Local area*	52-64	1,544	1,372 (89)
	65-74	909	755 (83)
	75-85	478	350 (72)
Total		2,940	2,477 (84)
Outside		· ·	. , , ,
local area	52-64	502	94 (19)
	65-74	357	78 (22)
- 4	75-85	178	26 (15)
Total	- 7	1,037	198 (19)

^{*}Includes all subjects living within a 25-mile radius of Framingham.

ation even when differences within the separate age groups are combined.

At age 52 to 64 years, the prevalence rate of diabetic retinopathy (Table 2) is about 2%, rising to about 3% at age 65 to 74 years, and 7% at age 75 to 85 years. The upward trend with age is significant (P < .01), but the differences by sex are not significant.

The overall prevalence of senile macular degeneration is 9% with prevalence rates of 2%, 11%, and 28% for the three age groups (Table 2). The increase with age is significant (P<.01), as are the higher rates for women as compared to men (P<.05).

The overall prevalence of open-angle glaucoma is about 3%. Prevalence increases with age (P<.01) from 1.4% at age 52 to 64 years, to 5.1% at age 65 to 74 years, and 7.2% at age 75 to 85 years. (Table 2). Rates for men are much higher than rates for women, and this difference is significant (P < .05).

DISCUSSION

Prevalence findings are always a function of the population under study and the potential bias related to nonresponse. The population chosen for this study is a rates than men, but the differences are subset of the adult population resident in Framingham since 1949, with a certain

	No. in Population	No. Screened	Senile Cataract or Aphakia (%)	Diabetic Retinopathy (%)	Senile Macular Degeneration (%)	Open-Angle Glaucoma (%)
Total	2940	2477	15.6	3.1	8.8	3.3
Men	1220	1043	13.5	3.0	6.7	4.1
Women	1720	1434	17:1	3.2	10.3	2.7
Age 52-64 yrs	1544	1293	4.6	2.1	1.6	1,4
Men	675	573	4.4	2,4	1.2	1.7
Women	869	720	4.7	1.9	2.0	1.2
Age 65-74 yrs	6 909	787	18.1 ·	2.9	11.0	5.1
Men	371	318	16.3	3.1	8.8	6.4
Women	538	469	19.3	2.7	12.6	4.3
Age 75-85 yrs	487	397	46.1	7.0	27.9	7.2
Men	174	. 152	41.5	5.2	24.4	9.9
Women	313	345	48.9	8.1	30.1	4.9

^{*}Table entries are the percentage of subjects screened positive for the condition in one or both eyes; local area residents only.

degree of selection by survivorship. Since 84% of the surviving heart study population residing in the greater Framingham area was examined, we have a reasonable basis for discussing prevalence. Little or no difference was found in the prevalence rates of individuals living within the Framingham area in comparison to those living outside it.

Epidemiological information about cataract came from three sources: (1) statistics of blindness registration, (2) population surveys, and (3) data derived from cataract extraction. Sorsby⁶ reported that senile cataract was the cause of 22% of blindness registrations at all ages in England and Wales for the years 1955 to 1962. In Sorsby's study, the proportion of registrable blindness caused by senile cataract increased with age and there was a greater frequency in women than in men.

Kornzweig, Feldstein, and Schneider⁷ studied all residents of a nursing home in New York City, composed of 400 men and 700 women over the age of 65 years. These authors excluded cases of incipient cataract with visual acuity of 6/12 (20/40) or better. They found that 31% of eyes among persons 65 to 79 years had

cataracts as defined, a prevalence level significantly higher than the prevalence of 18% found at Framingham, for the age group of 65 to 74 years. Although only a small number of nursing home residents were examined in the Framingham Eye Study, these individuals may have had a higher rate of ocular disease than that of the general population. Seemingly, individuals requiring nursing home care are physiologically older than their noninstitutionalized peers.

Milne and Williamson⁸ studied a random sample of older people in Edinburgh, comprising about 200 men and 300 women age 62 years or over, and found that 124 had vision of 6/9 (20/30) or worse, resulting from senile cataract. The Edinburgh survey showed a cataract prevalence of 22% for age 62 to 79 years, a figure similar to our Framingham finding of 18%. Comparable figures were obtained in a geriatric assessment survey in East Kilbride, a town of 70,300 with 6.5% of the population over age 65 years. Four thousand three hundred eves were examined and showed a total prevalence of cataracts of 14% for age 65 to 74 years and 10% for age 75 years and over. By contrast, Chatterjee¹⁰ examined over 20,000

people in the dry belt of the Punjab in India and found a cataract prevalence of 22% at age 50 to 59 years; this rate is considerably higher than the 4.6% found for the 52- to 64-year-old Framingham residents. The role of climatic, nutritional, and genetic factors in the higher prevalence of cataract in the dry belt of the Punjab is not certain. Nevertheless, the available data suggest the prevalence rates for cataracts in Framingham are comparable to those in Edinburgh and East Kilbride, and lower than those in the Punjab and the New York nursing home.

Although numerous data are available on the incidence of registrable blindness caused by diabetic retinopathy,11 no data are available on the prevalence of diabetic retinopathy in a general population. Knowles, Meinert, and Prout¹² estimate that the overall prevalence rate varies from 1.45% to 1.51% based on National Health Interview surveys. A rough estimate of retinopathy among the ... general population can be made by multiplying the figures for the prevalence of retinopathy among diabetics by the prev--alence of diabetes in the general population. Based on the 4,076 observations made at the Radcliffe Infirmary Diabetic Clinic between 1949 and 1965, one can estimate the prevalence of diabetic retinopathy in a general population of 1.1% between ages 50 to 59 years, 1.9% between ages 60 to 69 years, and 1.3% for _age 70 years and over. Analysis of the figures from the Joslin Clinic obtained by Kahn and Bradley¹³ gives 1.9%, 2.8%, and 3.0% as estimates of prevalence of retinopathy in the general population at ages 55 to 64 years, 65 to 74 years, and 75 years and over, respectively. The present study finds prevalence of retinopathy of 2.1%, 2.9%, and 7.0% at ages 55 to 64 years, 65 to 74 years, and 75 to 85 years, respectively. The Framingham data, at least in the younger age groups, are comparable to the figures calculated from the Joslin Clinic data.

Prevalence rates for senile macular degeneration are not readily available. Fülle¹⁴ found an incidence of 2.1% in the population seen in the Berlin University Eye Clinic. Kornzweig, Feldstein, and Schneider, in the nursing home study, claimed the prevalence of bilateral senile macular degeneration was 9% for those under age 80 years and 16% for those 80 years or over. Our study showed a prevalence of under 4% at age 65 to 74 years and under 20% at age 75 to 85 years. However, these data are not truly comparable because the authors7 failed to define criteria used to diagnose senile macular degeneration.

The estimated prevalence of open-angle glaucoma, one of the most common causes of blindness in people over the age of 40 years, varies from 5 to 40 per 1,000 in that age group. 15 Most of these prevalence data are suspect because we lack a standard definition of the disease. Ophthalmologists use three major criteria: (1) increased intraocular pressure, (2) increased cup:disk ratio, and (3) visual field loss. Often only one or two of the criteria are used, thus limiting comparison of results from different surveys. One of the best studies is by Björnsson,16 who surveyed 17 to 18% of the total population of Iceland, a racially homogenous country. Of the 27,715 Icelanders screened, 2% of the total population over age 40 years had clinical glaucoma as defined by IOP of 22.4 mm Hg (3/5.5 Schiøtz) or higher, with perimetric changes of Seidel's scotoma, Bjerrum scotoma, general constriction of visual field, the final stage of loss of central vision with remaining temporal island or field, or total blindness. Reports from two large eye clinics in the United States, the Massachusetts Eye and Ear Infirmary¹⁷ and the Wills Eye Hospital,¹⁸ described prevalence rates of 1.33% and 0.78% respectively, while Posner and Schlossman¹⁹ stated a 3.1% prevalence out of 12,000 patients who consulted them personally. In the studies of Hollows and Graham²⁰ and Wallace and Lov- als underwent an ophthalmologic evaluation that stressed detection of senile cataract, diabetic retinopathy, open-angle glaucoma, and senile macular degeneration. Those examined were 52 to 85 years old at the time this study was initiated. The prevalence rate of each of these ocu-

lar conditions increased with age.

Prevalence of senile cataracts ranged from 4.6% for those between the ages of 52 to 64 years to 46% for those 75 to 85 years of age. Diabetic retinopathy was. of primary glaucoma, based on abnormal present in 2% of those between 52 and 64 years of age or older. Overall prevalence of senile macular degeneration was 9%, with a prevalence rate of 2% in our youngest age group and 28% in the oldest age group. Open-angle glaucoma had an overall prevalence of approximately 3%. This disease also showed a statistically significant (P<.01) increase with age from 1.4% (52 to 64 years old) to 7.2% (75 to 85 years old).

ell,²¹ accurate comparison is possible, as both used common techniques and criteria for estimating IOP and optic disk changes. In the former study, of a population of predominantly Welsh origin, a prevalence of 0.47% of open-angle glaucoma was noted, while in the latter study, of a Jamaican population of West African Negro origin, a prevalence of 1.4% was noted. In the Bedford Glaucoma Survey, Bankes and associates²² detected 55 cases disks and a pressure of 21 mm Hg or more by applanation, among 5,941 persons. over the age of 40 years, with an overall prevalence rate of 0.93%. While our results of 3% prevalence for ages 52 years and above are definitely higher than the Icelandic, Welsh, West Indian, and Bedford surveys, they are comparable to Posner and Schlossman's figures.

Some of the observed differences between the Framingham data and those of other studies can be ascribed to the methods of examination and the definitions of disease. For example, senile macular degeneration in this study was defined as pigment abnormalities of the macula associated with a visual acuity of 6/9 (20/30) or worse; using this definition, we excluded minimal changes in macular pathology. Conversely, approximately one third of our glaucoma patients had blind spot enlargement as their only visual field abnormality. Some would not consider this change sufficient to make the diagnosis.

We expected the increasing frequency of these ocular disorders with age. What has not been properly emphasized in the past are the sex differences found in this and other studies. Senile macular degeneration occurs more frequently among women, and the prevalence of chronic simple glaucoma is higher among men.

SUMMARY

Of the Framingham, Massachusetts Heart Study population, 2,675 individu-

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MEASUREMENT OF EPISCLERAL VENOUS PRESSURE

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Diseases that elevate episcleral venous pressure include carotid-cavernous fistula, low-flow fistula between meningeal arteries and the cavernous sinus, 1 tumors obstructing the superior vena cava, and Sturge-Weber disease.2 Early diagnosis and treatment may, in some instances, forestall the serious adverse effects of these uncommon diseases. Ocular signs of elevated episcleral venous pressure include engorged episcleral vessels, elevation of intraocular pressure (IOP), and reflux of blood into Schlemm's canal that is visible during gonioscopy. Measurement of episcleral venous pressure facilitates recognition of these diseases and helps the ophthalmologist distinguish them from other causes of ocular hyperemia such as local irritation, conjunctivitis, scleritis, episcleritis, and orbital inflammation.

No instrument is available commercially for the measurement of episcleral venous pressure. Many instruments previously described for this purpose are expensive to build or difficult to use. Consequently, few clinicians are equipped to measure episcleral venous pressure.

For the past ten years we have mea-

sured episcleral venous pressure with a simple homemade instrument that provides rapid and reproducible measurements. We constructed the instrument easily from readily available, inexpensive materials. We used a modification of the classical pressure chamber method first used in 1880 by Roy and Brown³ to measure vascular pressures in the web; tongue, and mesentery of frogs and in the tails of newts and small fish. The pressure chamber method consists of placing a small transparent chamber, covered at . one end by a transparent membrane,. against the vessel to be measured, and increasing pressure within the chamber until the vessel collapses.

Several investigators,4-12 beginning with Seidel⁴ in 1923, have used pressure chamber instruments to measure episcleral venous pressure. None of their instruments has gained wide acceptance. Often the materials recommended for the membrane (intestinal serosa,4,7 latex films,6,8 softened cellophane,5 and pericardium of toads and frogs11,12) were difficult to prepare. Some instruments were filled with water or saline, thus adding to the inconvenience of preparation. The present instrument, which is similar to one described by Stepanik,9 overcomes these difficulties by utilizing a readily available membrane, latex Tonofilm, and an air-filled pressure chamber.

This report describes the construction and use of this instrument, the results of an investigation of episcleral venous pressure in normal subjects, and the clinical value of the measurement.

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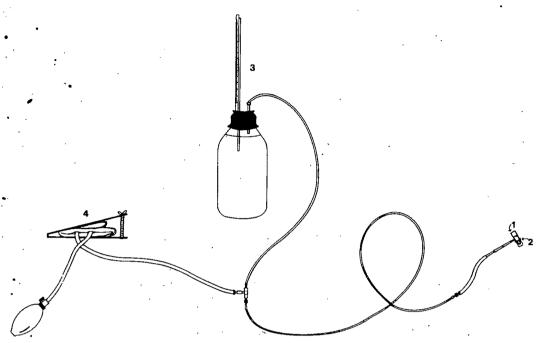


Fig. 1 (Phelps). Instrument for measuring episcleral venous pressure: 1, pressure chamber; 2, membrane; 3, manometer; and 4, device for varying the pressure.

MATERIAL AND METHODS

The instrument consists of four parts: (1) a pressure chamber; (2) a membrane; (3) a manometer; and (4) a device for varying the pressure within the instrument (Fig. 1). Flexible plastic tubing connects the various parts.

We constructed the pressure chamber by drilling out the small end of a Goldmann applanation tonometer prism, thus—converting it into a hollow plastic chamber open at one end and covered by transparent plastic at the other. The external diameter of the open end was 7 mm. A short length of stiff plastic tubing, glued into a hole drilled in one side of the chamber, served both as a handle and an attachment for flexible tubing leading to the manometer.

The membrane was tied over the open end of the chamber. Ideally, the membrane should be transparent, thin, elastic, soft and nonrigid, resistant to tearing, air tight, nonirritating, easily obtained, and easily attached to the instrument. We have tried over 25 different membranes. The least rigid, but most difficult to prepare, was toad pericardium. The most convenient was the latex Tonofilm, manufactured as a sterile Schiøtz tonometer cover. Although less transparent and stiffer than toad pericardium, the Tonofilm provided comparable results to pericardium in paired measurements. We easily fastened it over the end of the pressure chamber with a small rubber band (Fig. 2).

The manometer consisted of a bottle partially filled with water and closed tightly by a two-holed rubber stopper. Two rigid tubes were inserted into the stopper. One tube, which ended in the airspace above the water, was connected by flexible tubing to the pressure chamber. The other, a long tube extended

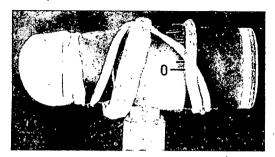


Fig. 2 (Phelps) Inflated latex membrane attached to pressure chamber by rubber band.

below the surface of the water, was marked at centimeter intervals, with 0 marking water level in the bottle.

To vary pressure within the system, we used an ordinary sphygmomanometer. This was connected to a side arm of the tubing leading from the pressure chamber to the manometer. The cuff of the sphygmomanometer was rolled and placed between two metal plates hinged along one edge and connected by a screw at the other. The insufflation bulb was used for coarse adjustments. For fine adjustments, we tightened or loosened the screw, thus either squeezing or releasing the partially inflated cuff.

The manometer bottle can be set at any level in relation to the pressure chamber without affecting the measurement. The reading in centimeters of water is easily converted to millimeters of mercury by multiplying it by a conversion factor of 0.74.

We performed measurements by using the slit-lamp biomicroscope for illumination and magnification. A green filter reduced glare and increased contrast between the blood column and underlying sclera. Measurements were most conveniently made in the temporal or nasal sectors of the perilimbal sclera. Topical anesthesia was used.

While the membrane surface of the

pressure chamber was alternately applied to and lifted from the eye, we observed the underlying vessels for color, caliber, and continuity. The chamber should be applied firmly, so that the area of membrane applanation goes slightly beyond the vessel being studied. However, the plastic rim of the chamber should neither touch the eye nor press on the eyelid. Because the vein was occluded for only short intervals, a damming-up effect was avoided.

Initially, the chamber pressure was raised above venous pressure, thus allowing the observer to distinguish veins from arteries. Pressure in the chamber was then lowered or raised as needed, usually in increments or decrements of about 1 cm H₂O. Observations were made at each pressure level, with the observer taking ample time to convince himself of the presence or absence of venous collapse.

There may be a difference of 2 to 3 mm. Hg between the pressure required to slightly indent the blood column and that required to completely obliterate the vessel. Between these levels, the blood column gradually blanches. The first variations in intensity of color are subtle and difficult to reproduce consistently. On the other hand, complete obliteration of the blood column requires too high a pressure. The vein, which is elliptical in cross-section, collapses first in its midportion. Rigidity of its walls prevents its complete collapse until the external pressure is raised considerably above intraocular pressure. It is easy to determine a sharp end point, which is, at most, 0.05 to 1.00 cm H₂O below the first observable changes, and is reproducible. At this point the blood column is still approximately its original width, but is blanched. A slight further increment in pressure obliterates part of the column.

Selection of a proper vein is important. Conjunctival veins are readily identified

because they move over the underlying tissues. Episcleral veins are fixed to the underlying sclera. Veins of similar caliber and location should be measured in each eye of the patient.

Episcleral veins have a higher pressure if measured immediately adjacent to the point where they emerge from the sclera than if measured at a point several millimeters "downstream." This drop in pressure may be real or it may be an artifact caused by the oblique course of the vessel through the sclera. At the point where the vessel first becomes visible, it is still partially intrascleral and thus partially protected by the sclera from the pressure of the chamber. Greater pressure is required to collapse the vein, and a falsely high estimate results.

To check the reliability of the instrument, we measured episcleral venous pressure three times in each eye of 28 normal individuals. Ages of the subjects were evenly distributed between 18 and 75 years. Measurements were read from the manometer scale to the nearest 0.5 cm H₂O and then converted to millimeters of mercury. Each measurement was masked in that the examiner was unaware of the pressure level in the manometer until he had decided on the end point. We recorded the average of the three measurements in each eye as the episcleral venous pressure of that eye.

RESULTS

Normal Subjects—The range between the highest and lowest of the three measurements in each eye varied from 0.4 to 2.3 mm Hg. It averaged 1.1 ± 0.5 mm Hg (mean \pm S.D.). The variation between the three measurements was less than 1 mm Hg in 25 eyes, 1 to 2 mm Hg in 27 eyes, and over 2 mm Hg in only four eyes.

The average episcleral venous pressure for the 56 eyes was 9.0 ± 1.6 mm (mean \pm S.D.). The lowest pressure recorded

TABLE 1

Case 1.

Effect of low-flow arteriovenous fistula
on intraocular pressure, episcleral
venous pressure, and exophthalmos

Date	Intraocular Pressure (mm Hg) R.E. L.E.	Episcleral Venous Pressure (mm Hg) R.E. L.E.	Exophthalmometry (mm) R.E. L.E.
11/12/75 12/ 1/75 12/16/75 1/20/76 3/ 5/76 4/23/76 8/24/76	10 24 12 22 11 21 12 28 12 20 11 17 13 14	10 18 9 16 8 21 — — — — 7 8	17 23 15 23 16 26 17 23 17 22 16 18

was 5.6 mm Hg and the highest was 12.0 mm Hg. Average pressure for right eyes was 9.2 mm Hg and for left eyes was 8.8 mm Hg. The difference between the two eyes was more than 2 mm Hg in two subjects, between 1 and 2 mm Hg in six subjects, and less than 1 mm Hg in 20 subjects.

CASE REPORTS

Case 1—A 60-year-old man complained of left-sided headache, double vision, and sudden proptosis of his left eye. Examination revealed a normal right eye. The left eye had a superior oblique paresis, 6-mm exophthalmos, prominent episcleral vessels, and gonioscopy revealed blood filling Schlemm's canal. Intraocular and episcleral venous pressures were elevated in the left eye, but not in the right (Table 1). Outflow facility was normal in each eye (0.28 μl/mm Hg/min in the right eye, and 0.26 μl/mm Hg/min in the left eye).

Orbital echography revealed a large pulsating vein in the left orbit. Doppler studies demonstrated reversal of venous flow. Carotid angiograms showed a low flow arteriovenous fistula filling the basilar venous plexus, the cavernous sinus, and the petrosal sinus and ophthalmic veins. We presumed the shunt came from one of the small branches of the meningohypophyseal artery.

No treatment was given. Signs and symptoms remained the same for about three months and then slowly resolved. Episcleral venous pressure returned to normal. The patient remains well one year later. We assume that a small arteriovenous fistula closed spontaneously.

Case 2—A 21-year-old woman said her right eye had been red for one month. Eighteen months

earlier she had undergone surgery for a chemodectoma involving the right pterygoid space, base of skull, and middle cranial fossa. Recovery from this extensive operation was uneventful and complete, but 12 months later (five months before her eye became red) she was in an automobile accident which resulted in a right sixth nerve palsy.

Examination revealed marked hyperemia of the right epibulbar vessels. Intraocular pressure was R.E.: 18 mm Hg, and L.E.: 11 mm Hg. Episcleral venous pressure was R.E.: 16 mm Hg, and L.E.: 9 mm Hg. Outflow facility was R.E.: 0.24 μl/mm Hg/min, and L.E. 0.27 μl/mm Hg/min. Caroud angiography revealed a right carotid-cavernous fistula Through a craniotomy incision, the right oph- thalmic and external carotid arteries were clipped. A muscle embolus was used to plug the right internal carotid artery.

Ten days later, IOP was 14 mm Hg in each eye; episcleral venous pressure was R.E.: 7.5 mm Hg, and L.E.: 8 mm Hg. She remains well one year later.

Case 3-A 58-year-old man, a heavy smoker, complained of shortness of breath. He had periorbital edema and distended cervical veins. Chest roentgenograms revealed a large density containing an air fluid level in his right upper lobe. Bronchoscopy revealed a large mass occluding the right mainstem bronchus. Biopsy showed the mass to be a bronchogenic carcinoma.

Intraocular pressure was 20 mm. Hg in each eye, and episcleral venous pressure was 16 mm Hg in each eye. A diagnosis of superior vena caval obstruction was made. Irradiation treatment caused temporary amelioration of symptoms. Episcleral venous pressure decreased to R.E.: 11 mm Hg, and L.E.: 12.5 mm Hg. He died soon afterward of complications of metastatic carcinoma.

Case 4-A 54-year-old man was referred for measurement of episcleral venous pressure because of dilated episcleral vessels in his right eye. The right eye also had reduced visual acuity and occlusions of several branch retinal arterioles. Arteriovenous fistula was considered as a possible cause.

However, episcleral venous pressures were 8 mm Hg in both eyes, and IOP was 14 mm Hg in both eves. Further examination revealed the episcleral vascular dilation was confined to the interpalpebral region. He had worked for much of his life shovelling coal into a furnace, with the right side of his body turned toward the fire. Subsequent investigations revealed severe carotid artery occlusive disease as the presumed cause of his retinal vascular disease. The episcleral vascular dilation was probably caused by chronic exposure of the right eye to high heat.

Case 5-During a routine ocular examination, a 12-year-old boy was discovered to have high IOP in his left eye. Since birth he had had a hemangioma (port-wine stain) involving the left side of his face, including the left upper eyelid.

Visual acuity was 6/6 (20/20) in each eye, but perimetry revealed a nasal step in the left visual field. A hemangioma involved the episcleral vessels in his left eye, and the left cornea was 1 mm larger than the right cornea. The left optic disk had glaucomatous cupping. The chamber angle had a normal configuration, but blood refluxed into Schlemm's canal during gonioscopy. Intraocular pressure was R.E.: 15 mm Hg, and L.E.: 35 mm Hg. Episcleral venous pressure was R.E.: 8 mm Hg, and varied from 18 to 16 mm Hg in the left eye. We recorded the highest pressures from small episcleral veins in the hemangioma; lower pressures were recorded from large episcleral veins some distance from the hemangioma. A diagnosis was made of Sturge-Weber disease with glaucoma secondary to elevated episcleral venous pressure. The elevated episcleral venous pressure probably resulted from arteriovenous shunting in the episcleral hemangioma.

DISCUSSION

Episcleral venous pressure is worth. measuring for two reasons: (1) it is one of . the physiological factors which determines IOP; (2) its elevation can be accurately measured and is a cardinal sign of several diseases that obstruct drainage of venous blood from the orbit. The episcleral veins are like a physiologic "sink" that aqueous humor empties into as it leaves the eye through the trabecular meshwork; Schlemm's canal collector channels, and aqueous veins. Episcleral venous pressure is independent of variations in IOP; however, IOP is not independent of episcleral venous pressure. The dependence of IOP on episcleral venous pressure is clarified by the familiar rearrangement of Goldmann's classical model of aqueous outflow:

$$P_0 = F/C + P_{ev}$$

 $P_{\rm o} = F/C + P_{\rm ev}$ According to this formula, a rise in episcleral venous pressure (P_{ev}) , unless compensated by a fall in the rate of aqueous flow (F) or an increase in outflow facility (C), will cause an equal rise in IOP.

Pressure in the episcleral veins is determined by body position and by the tissue pressure of the orbit, head, and neck. If a subject is sitting or standing, pressure in the episcleral veins is maintained by force transmitted from the arteries through the episcleral capillaries at a level just high enough to prevent collapse of the ophthalmic and jugular veins by the extramural tissue pressure. In a recumbent or head dawn position, the subject's episcleral venous pressure is also determined by how far below the level of the right atrium the eye is.

Because tissue pressure in the orbit, head, and neck probably undergoes little spontaneous variation, the role of episcleral venous pressure in determining the level of IOP is normally static and comes into play only with changes in body position. Few varieties of glaucoma are caused by abnormalities of episcleral .venous pressure^{8,10,16}; the high IOP in · most types of glaucoma results from reduction of outflow facility. In a few diseases, however, including arteriovenous fistulas, superior vena cava obstruction, and Sturge-Weber disease, high episcleral venous pressure causes increased IOP. Direct measurement is the most accurate way of proving elevated episcleral venous pressure to be a cause of secondary glaucoma. Patients with prominent episcleral vessels or spontaneous reflux of blood into Schlemm's canal may have high episcleral venous pressure, but actual measurement must confirm it:

The first four case histories illustrate the value of episcleral venous pressure measurement in the diagnostic evaluation of a patient with a red eye. In the first three patients, measurement of episcleral venous pressure not only confirmed obstructed venous drainage, but also provided a useful means of assessing the response to therapy or the natural course of the disease. In the fourth case, the measurement proved the patient's red eye was not caused by elevated episcleral venous pressure. The fifth case illustrates how measurement of episcleral venous pressure can be used to elucidate disease mechanisms. (The role of elevated episcleral venous pressure in the pathogenesis of Sturge-Weber disease is discussed in detail elsewhere.²)

The present instrument provides reproducible measurements of episcleral venous pressure. Our results in normal subjects are similar to those obtained by other investigators using a variety of techniques and instruments (Table 2). 4-10,12-18 Our instrument is easy to use and allows for quick measurements. Preparation for each measurement consists only of fastening a new latex membrane to the pressure chamber. The entire measurement, including preparation and replicate measurements, takes less than five minutes.

The accuracy of any pressure chamber instrument has at least one inherent limitation. The technique assumes that collapse of a vein does not occur until extravascular pressure (the pressure within the chamber) exceeds intravascular pressure. This assumption may not be completely correct. If active tension caused by smooth muscle contraction is present in the wall of a vein, the vein may collapse while the transmural pressure is still slightly positive. Because episcleral veins have smooth muscle and adrenergic innervation, they may have active tension in their walls. Thus, the estimate of episcleral venous pressure obtained by the pressure chamber technique may be slightly low. The magnitude of this effect, how it varies from subject to subject, and how it is affected by topically applied medications are unknown.

`SUMMARY

Using materials available in any ophthalmology clinic, we constructed a useful and reliable instrument for measuring epischeral venous pressure. The instrument, a modification of the pressure chamber method of Seidel, utilizes a latex membrane and an air-filled chamber.

TABLE 2
RESULTS OF STUDIES ON EPISCLERAL VENOUS PRESSURE IN NORMAL EYES

Authór	No. of Eyes	Episcleral Venous Pressure (mm Hg) (Mean ± S.D.)	Method
Seidel ⁴	?	Range: 7-11	Pressure chamber with serosal membrane
Thomassen ⁵	3	Range: up to 65	Pressure chamber with softened cellophane
Linnér ⁶	24	14.3 ± 1.02	Pressure chamber with rubber membrane
Goldmann ¹³	20	9.7 ± 2.2	Torsion balance
Rickenbach and Werner ¹⁴	10	11.4 ± 1.5	Torsion balance
Linnér, Rickenback,	38	10.0 ± 2.6	Torsion balance
and Werner ¹⁵	38	11.4 ± 1.4	Pressure chamber with rubber membrane
Weigelin and Löhlein ⁷	103	9.7 ± 2.5	Pressure chamber with serosal membrane
Ĺinnér ⁸	28	11.0 ± 1.4	Pressure chamber with rubber membrane
Stepanik ⁹	25	19.1 ± 7.2	Pressure chamber with rubber membrane
Leith ¹⁰	20	10.4 ± ?	Pressure chamber with rubber or plastic membrane
Kupfer and Sanderson ¹²	21	8.3 ± 1.1	Pressure chamber with frog pericardium for membrane
Podos, Minas, and Macri ¹⁶	39	9.0 ± 1.4	Force-displacement transducer
Stepanik ¹⁷	16	10.4 ± 4.1	Applanation measurement of intraocular pressure after prolonged
Krakau, Widakowich, and Wilke ¹⁸	4	Range, 14.3-20.1	indentation tonometry Air jet
Present study	5 6	9.0 ± 1.6	Pressure chamber with rubber membrane
			•

These modifications facilitated ease of preparation for the measurement. Episcleral venous pressure in normal subjects was 9.0 ± 1.6 mm Hg (mean \pm S.D.). Measurement of episcleral venous pressure facilitated diagnosis of diseases such as arteriovenous fistula and superior vena caval obstruction, which block drainage of venous blood from the orbit.

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OPHTHALMIC MINIATURE

To Err Is Human—The Book Review is now set in cold type by computers, and locally written columns such as this one are typed out on the keyboards of Video Display Terminals, the words produced not by metal, ink and paper but by electronic impulses on a screen. The process makes us all wonderously faster and more efficient. Still, the misspelling of T.S. Matthews's name two weeks ago was a human error. Human beings, you see, remain in charge here. New York Times, December 11, 1977.

PHENYLEPHRINE PROVOCATIVE TESTING IN THE PIGMENTARY DISPERSION SYNDROME

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The pigmentary dispersion syndrome is characterized by a loss of pigment from the posterior iris surface in the midperiphery, and its subsequent deposition on intraocular structures such as the cornea, trabecular meshwork, iris, and lens.1-5 The condition results in a striking transillumination pattern of the iris, 2,3,5,6 as well as a Krukenberg spindle, trabecular pigment band, subtle heterochromia, and posterior lenticular pigmentation. It has not been adequately stressed that the pigmentary dispersion syndrome may occur with or without glaucoma (pigmentary glaucoma). We have often observed the full dispersion syndrome without glaucoma in otherwise normal patients.

Sugar⁴ believes that elevated intraocular pressure is a late-appearing component of the full syndrome, and he has observed the onset of glaucoma 12 to 20 years after pigmentary dispersion was first detected.⁷ Nevertheless, not all patients with pigmentary dispersion syndrome develop glaucoma even after 20 years of follow-up (Case 1). A diagnostic test to identify latent pigmentary glaucoma in patients with the dispersion syndrome would be useful.

Phenylephrine mydriasis may result in the liberation of pigment granules into the aqueous humor.⁸⁻¹⁰ These pigment particles are believed to originate from posterior iris surface neuroepithelial cells, which rupture during iris dilator · muscle contraction.8 Ordinarily phenylephrine either reduces or does not alter intraocular pressure in normal persons and open-angle glaucoma patients.9-12 Rare cases of paradoxical pressure rise, despite open angles, have been reported following phenylephrine mydriasis. 13,14 Kristensen¹⁵ reported a marked rise in intraocular pressure associated with excessive liberation of pigment into the anterior chamber after phenylephrine mydriasis in patients with pigmentary glaucoma and pseudoexfoliative glaucoma. He believed the mechanism of the pressure rise to be temporary blockage of the trabecular meshwork by the liberated. pigment particles.15

We wished to learn the incidence of such a phenylephrine-induced, pigment-associated pressure rise in patients with pigmentary glaucoma, and to determine whether this test could identify latent glaucoma in patients with the pigmentary dispersion syndrome. Additionally, we documented two cases of pigmentary glaucoma in which either spontaneous or exercise-induced liberation of pigment into the anterior chamber was associated with a marked rise of intraocular pressure and obstruction of aqueous outflow.

SUBJECTS AND METHODS

We studied 49 patients with pigmentary dispersion syndrome or pigmentary glaucoma. All patients had had slit-lamp examinations, gonioscopy, iris transillumination, optic disk drawings (often with stereophotographs), tonography, and visual fields as part of their routine examinations here. The first four of these examinations were repeated on all patients (by

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D.L.E. and W.P.B.) before phenylephrine provocative testing.

We diagnosed cases of pigmentary dispersion syndrome, with or without glaucoma, by observing the presence of the characteristic peripheral iris transillumination pattern^{2,3,5,6} and a dense pigment band in the trabecular meshwork, and the absence of exfoliation, cysts, or melanoma of the iris or ciliary body. The presence of the characteristic iris transillumination pattern was considered essential to the diagnosis of pigmentary dispersion syndrome. Patients whose irides did not transilluminate, but who had dense trabecular pigment bands with corneal endothelial pigmentation (Krukenberg spin-·dles) without other cause were listed as having "probable" pigmentary dispersion syndrome. Patients with moderately dense trabecular pigment bands who had abnormal iris transillumination and other signs of pigmentary dispersion were also listed as probable pigmentary dispersion syndrome cases. These patients would have been classified as having pigmentary dispersion syndrome according to previously published criteria.5

We diagnosed pigmentary glaucoma, or

"pigmentary dispersion syndrome with glaucoma," when Goldmann applanation intraocular pressures were repeatedly 24 mm Hg or more and results of tonography were abnormal (C value of 0.15 or less). Patients both with and without glaucomatous disk damage and field loss were classified as pigmentary glaucoma cases on the basis of abnormal intraocular pressure and tonographic results, although they obviously represented different stages of the disease process. These cases would have been classified as pigmentary glaucoma according to previously published criteria.⁵ Pigmentary dispersion syndrome without glaucoma was diagnosed if repeated Goldmann applanation intraocular pressures were 21 mm Hg or less, and tonographic C values were 0.19 or more. As a subgroup of those with the syndrome but without glaucoma, patients with intraocular pressures of 22 to 23 mm Hg and C values of 0.16 to 0.18 were classified as pigmentary glaucoma "suspects."

For phenylephrine provocative testing, one drop of 10% phenylephrine was applied randomly to one eye of a pigmentary dispersion patient every five minutes.

TABLE 1

AGE AND SEX OF PATIENTS WITH PIGMENTARY DISPERSION SYNDROME
WITH AND WITHOUT GLAUCOMA

	Number	Average Age at Time of Testing (yrs)	Average Age at Time of Diagnosis (yrs)
Pigmentary glaucoma	b.		
Men	19	44	41
Women	12	54	48
Pigmentary glaucoma suspects	*		
Men	4	43	38
Women	3	44	40
Pigmentary dispersion without glaucoma			
Men	10	44	40
Women	8	48	45
Total	-		
Men	29	44	41
 Women 	20	52	. 47

three times. The fellow eye with pigmentary dispersion syndrome served as a control. Applanation pressure, anterior chamber pigment floater reaction, and pupillary diameter were recorded in a darkened room both before and between 60 and 120 minutes after phenylephrine administration. Anterior chamber pigment floater reaction was graded according to a modified Mitsui⁸ scale in which grades 1+ to 4+ in our study correspond to Mitsui grades 3 to 6. No attempt was made to alter or to discontinue topical antiglaucoma therapy before phenylephrine testing because the pressure rise with phenylephrine has occurred in patients both on and off topical therapy. 15

Iris transillumination was performed, after dark adaptation to a darkened room, with a fiber optic transilluminator applied to the lower eyelid or sclera. Grading was: "minimal" when one to four individual peripheral slits showed; "moderate" when less than 180 degrees of the circumference transilluminated; "extensive" when more than 180 degrees of the circumference transilluminated.

Pearson regression lines were calculated on a computer calculator.

RESULTS

We recorded the age and sex of the 49 patients with pigmentary dispersion syndrome (Table 1). Seventeen of 31 (55%) patients with pigmentary glaucoma, and

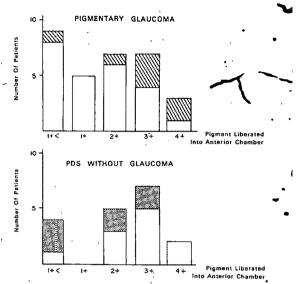


Fig. 1 (Epstein, Boger, and Grant). Top, Incidence of different grades of phenylephrine-induced pigment liberation into the anterior chamber in pigmentary glaucoma. Cross-hatched areas represent cases classified as "probable" pigmentary glaucoma. Bottom, Incidence of different grades of phenylephrine-induced pigment liberation into the anterior chamber in pigmentary dispersion syndrome without glaucoma. Dotted areas represent patients classified as pigmentary glaucoma "suspects."

14 of 18 (78%) patients with pigmentary dispersion syndrome without glaucoma, demonstrated a 2+ or greater grade of pigment liberation in response to phenylephrine (Fig. 1). Within both groups was a tendency for greater incidence of such pigment liberation with increased age (Table 2). Overall, six of 14 (43%) pa-

TABLE 2

PROPORTION OF PATIENTS DEVELOPING 2+ OR GREATER ANTERIOR
CHAMBER PIGMENT LIBERATION IN RELATION TO PATIENT AGE

Age (yrs)	Pigmentary Glaucoma	Pigmentary Dispersion Without Glaucoma	Total (%)
10-29	3/3	1/3	4/6 (66)
30-39	2/7	0/1	2/8 (25)
40-49	1/4	5/6	6/10 (60)
50-59	5/7	4/4	9/11 (82)
60-79	6/10	4/4	10/14 (7)

TABLE 3 INCIDENCE OF 24 OR GREATER ANTERIOR CHAMBER PICMENT LIBERATION IN RELATION TO IRIS TRANSILLUMINATION

Transillumination of the Iris	Pigmentary Glaucoma (%)	Pigmentary Dispersion Without Glaucoma (%)	Total (%)
Mînimal	4/8 (50)	3/4 (75)	7/12 (58)
Moderate	7/12 (58)	4/5 (80)	11/17 (65)
Extensive	6/11 (54)	7/9 (78)	13/20 (65)

tients with pigmentary dispersion syndrome under the age of 40 years demonstrated such pigment liberation, as compared to 25 of 35 (71%) patients who were older than 40 years (Table 2). Only five of 49 patients, three with glaucoma and two without, developed a 4+ pigment reaction (Fig. 1).

In patients both with and without glaucoma, there was no correlation between the extent of iris transillumination and the grade of phenylephrine-induced pigment liberation (Table 3). Patients with pigmentary glaucoma who were receiving miotic or epinephrine antiglaucoma therapy at the time of phenylephrine testing. demonstrated a higher incidence of the pressure rise revealed that the angles phenylephrine-induced pigment libera- had remained open. tion than those pigmentary glaucoma patients who were not on topical therapy at the time of the testing (Table 4).

There was a wide variation in the intraocular pressure response to the phenylephrine testing (Figs. 2 and 3). Only three of 31 pigmentary glaucoma patients and

one of 18 pigmentary dispersion patients without glaucoma (but this one was a pigmentary glaucoma suspect) developed a pressure rise greater than 2 mm Hg during the phenylephrine testing (Figs. 2) and 3). During this time, the fellow eye in these patients did not change more than ±1 mm. Hg. In patients who developed 3+ or 4+ pigment liberation, two of ten patients with pigmentary glaucoma manifested a greater than 2 mm Hg pressure rise, as compared to 0 of nine without glaucoma (Figs. 2 and 3). The highest pressure rise observed from phenylephrine in these 49 patients was 7 mm Hg (Fig. 2). Gonioscopy in the patients with

CASE REPORTS

'Case 1-The case of a 76-year-old white man was diagnosed as pigmentary dispersion syndrome in the emergency room where he sought treatment for blepharitis. According to records of his ophthalmologist, he had been observed for 20 years for heavy Krukenberg spindles in both eyes, with normal intraocular pressure; he received no treatment.

TABLE 4 INCIDENCE OF 2+ OR GREATER ANTERIOR CHAMBER PIGMENT LIBERATION IN RELATION TO TOPICAL ANTIGLAUCOMA MEDICATIONS

Therapy Pigmentary	Glaucoma	Pigmentary Without G	Dispersion laucoma
Epinephrine 15/2 (Epinephrine alone) (5/4 Miotics 11/2 None 2/2	6) 16	14/1	18

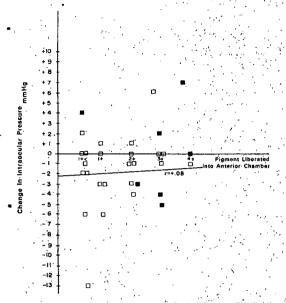


Fig. 2 (Epstein, Boger, and Grant). Effect of phenylephrine on anterior chamber pigment liberation and intraocular pressure in pigmentary glaucoma. Open squares represent pigmentary glaucoma. Solid squares represent "probable" pigmentary glaucoma as defined in the test. The computed trend is represented by the r (regression) line.

See . Examination confirmed the presence of broad Krukenberg spindles in both eyes. The anterior chambers were deep, with occasional pigment particles present spontaneously in the aqueous humor of the right eye; and 1+ spontaneous pigment particles in the left eye. No exfoliation was present and there was moderate pigment deposition on the anterior iris surface in both eyes. Marked iridodonesis was in both eyes. Striking, spoke-like, midperipheral iris transillumination was present in all quadrants in each eye. Applanation pressure was 19 mm Hg in each eye. Gonioscopy revealed wide open angles with dense continuous trabecular pigment bands in each eye. There was some dense uveal meshwork in both eyes which was not thought to represent abnormal "iris processes." ^{15,16} The disks showed healthy physiological cupping and the visual fields were full. Tonography C values were R.E.: 0.33; and L.E.: 0.38. Phenylephrine provocative testing of the right eye revealed an increase in the amount of anterior chamber pigment floater particles to 3+. with no change in intraocular pressure.

Case 2—A 30-year-old myopic white man noticed blurring of vision and halos after exercise, greater in the right eye than the left eye. Pressure was 40 mm Hg in both eyes. The patient was in good health with no family history of glaucoma.

Examination on another occasion, when the patient was asymptomatic, revealed diffuse deposits of pigment on the back surface of both corneas, and the shadow of a band of pigment in each angle showed

by retro-illumination through the sclera at the corneoscleral limbus. The anterior chambers were abnormally deep. Gonioscopy revealed an almost concave iris contour with a dense, black band of pigment 360 degrees trabecular meshwork in both eyes. Pigment was behind the equator of beth enses. The irides transilluminated in the midperiphery. The disks had small physiologic cups. Applanation pressures were R.E.: 20 mm Hg, and L.E.: 19 mm Hg. There was an occasional speck of pigment floating in the aqueous in both eyes.

The patient agreed to jog for two hours, to provoke his symptoms. When he returned, he reported mild onset of symptoms. Applanation pressures were R.E.: 31 mm Hg, and L.E.: 22 mm Hg. There were diffuse, fine clouds of pigment in the aqueous of the right eye, greater than in the left eye. Tonography C values after exercise were R.E.: 0.02, and L.E.: 0.05. Before exercise, tonography C for the right eye had been 0.09. The patient stated that activity in which he "jiggled" his head, such as basketball, caused more symptoms than simple running.

Seven months later the patient returned with symptoms of halos in the right eye. Intraocular pressures were R.E.: 38 mm Hg, and L.E.: 22 mm Hg with a great amount of circulating pigment in the anterior chamber of the right eye, and considerably less in the left eye.

Subsequently the patient returned for phenyleph-

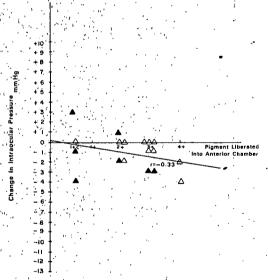


Fig. 3 (Epstein, Boger, and Grant). Effect of phenylephrine on anterior chamber pigment liberation and intraocular pressure in pigmentary dispersion syndrome without glaucoma. Open triangles represent pigmentary dispersion snydrome without glaucoma. Solid triangles represent those patients who were classified as pigmentary glaucoma suspects. The computed trend is represented by the r (regression) line.

rine provocative testing. After phenylephrine, the right eye showed only an occasional pigment speck in the anterior chamber with no significant change in tension, but an increase in facility to 0.14. Subsequent phenylephrine testing of the left eye revealed only in significant change in the anterior chamber with no significant change in tension or facility.

Case 3-In 1968 a 30-year-old white man with · pigmentary glaucoma gave a history of intermittent attacks of alternating monocular pain, halos, and blurred vision lasting three to four hours. He associated these attacks with dim illumination and occasionally with emotional circumstances, but not exercise. Intraocular pressures when he was asymptomatic were in the low 20s in the right eye, and low 30s in the left eve. Tonography C values were R.E.: 0.10, and L.E.: 0.06. The angles were open, of safe width, with dense trabecular pigment bands. No pigment particle floaters were noted in the anterior chambers. The disks had small physiological cups. Darkroom testing resulted in no pressure rise. The patient was treated with epinephrine and pilocarpine in the left eye for several years.

In 1975, after treatment had been discontinued, phenylephrine provocative testing was performed in the left eye. The intraocular pressure decreased from 34 to 21 mm Hg in the left eye, and only a rare pigment granule was seen in the anterior chamber of the left eye. The right eye remained unchanged at 24 mm Hg. The patient was observed further without

treatment.

In December 1976 he was seen during a spontaneous attack in the right eye that occurred while he was working outside in dim light at night. Intraocular pressure was R.E.: 55 mm Hg, and L.E.: 22 mm Hg. There were 4+ multisized pigment particle floaters in the anterior chamber of the right eye, and only occasional particles in the left eye. Krukenberg spindles were prominent in both eyes. Gonioscopy revealed both angles to be open with dense trabecular pigment bands. The patient was treated with acetazolamide and was asymptomatic the following morning.

Case 4—In a 75-year-old white woman under treatment for primary open-angle glaucoma, the corneas were clear, the angles were open without abnormal pigmentation, and the irides did not transilluminate. Tonography C values were R.E.: 0.14, and L.E.: 0.12. The disks had wide physiological cups that did not reach the rims, and fields were full. Under treatment with echothiophate, epinephrine, and acetazolamide the intraocular pressures were in the low 30s.

On five occasions phenylephrine testing was performed on one or the other eye and the intraocular pressure in the tested eye rose 10 to 27 mm Hg (mean 16 mm Hg) above the pretest pressure, into the upper 40s and low 50s. On four of these occasions the fellow untreated eye showed no rise in pressure, and once it rose 5 mm Hg. During this testing, a diffuse cloud of extremely fine pigment particles was observed in the anterior chamber of the treated eye. Gonioscopy revealed the angle was always open during these pressure elevations.

DISCUSSION

The basic abnormality in pigmentary glaucoma has been a subject of controversy. Several authors^{3,4,7,17} have suggested pigment obstruction of the aqueous outflow channels as a cause for the glaucoma, while others have ascribed the glaucoma to congenital mesodermal angle anomalies.5,16,18,19 Initial studies 20 of corticosteroid testing suggested that pigmentary glaucoma was a variant of primary openangle glaucoma, but more recent studies have suggested that these two diseases are distinct entities.21,22 Hyperplasia of the iris dilator muscle^{23,24}and degeneration of iris nerve elements24 have been observed in pigmentary dispersion syndrome, but the exact relationship of these iris abnormalities to the basic mechanism of the glaucoma has been uncertain.

Cases 2 and 3 support the possibility of at least a temporary obstruction of aqueous outflow channels by pigment particles in certain pigmentary glaucoma patients, although both the pigment liberation and elevated pressure may have been manifestations of some other process. Experimentally, pigment particles can obstruct aqueous outflow in perfused enucleated primate eyes, 25-27 and pigment particles have been observed "plugging" the intertrabecular spaces in pathological specimens of pigmentary glaucoma.28,29 Kristensen¹⁵ reported a phenylephrineinduced, pigment-liberation associated pressure rise in pigmentary glaucoma and also suggested that pigment particles are capable of obstructing the outflow channels.5

We observed that less than one third of patients with pigmentary glaucoma developed a 3+ or 4+ pigment response after phenylephrine administration (Fig. 1), and of these only 20% (two of ten) developed an appreciable intraocular pressure rise (Fig. 2). The highest pressure rise observed in the 31 pigmentary glaucoma patients was 7 mm Hg, which was considerably lower than that in

Kristensen's case, 15 and in our patient with primary open-angle glaucoma (Case 4). There was a higher incidence of significant pigment liberation in pigmentary dispersion syndrome patients with normal pressures than in glaucomatous patients (Fig. 1), but there was essentially no intraocular pressure rise associated with a 3+ or 4+ pigment response in the former (Fig. 3). There was a trend for a decrease in intraocular pressure with greater pigment liberation in nonglaucomatous pigmentary dispersion syndrome patients (Fig. 3).

The variation in pigment liberation and the low incidence of a pressure rise following phenylephrine administration suggest that the test will not be useful in. classifying pigmentary glaucoma patients or identifying latent glaucoma in patients with the pigmentary dispersion syndrome. Yet the aqueous pigment floater results remain puzzling. Perhaps the pressure-reducing action of phenylephrine itself9-12 obscured the pressureelevating effects of pigment liberation. More eyes had a decrease of pressure than a rise (Figs. 2 and 3). Despite this concomitant tendency of the drug to reduce pressure in some eyes, the rise in intraocular pressure that can be induced by phenylephrine supports the concept that pigment particles under certain circumstances temporarily obstruct the aqueous outflow channels.

SUMMARY

Forty-nine patients with bilateral pigmentary dispersion syndrome (abnormal accumulation of pigment in the anterior chamber, principally from the posterior layers of the iris), including 31 patients with pigmentary glaucoma, underwent 10% phenylephrine testing in one eye for evaluation of liberation of pigment floaters into the anterior chamber and the influence of phenylephrine on the intraocular pressure. Ten patients with pigmentary glaucoma developed a 3+ to 4+

pigment response, but only two demonstrated a pressure rise greater than 2 mm. Hg. The highest pressure rise observed was 7 mm Hg. Nine patients with pigmentary dispersion syndrome but without glaucoma also developed a 3+ to 4+ pigment response, but none of these had a pressure rise. The incidence of pigment liberation was higher in older patients and in pigmentary glaucoma patients receiving topical antiglaucoma therapy at the time of testing. The extent of iris transillumination did not correlate with the grade of phenylephrine-induced pigment liberation.

Two pigmentary glaucoma patients, who did not liberate pigment or have a pressure rise when tested with phenylephrine, did exhibit spontaneous or exercise-induced liberations of pigment into the anterior chamber, with marked rises of intraocular pressure and obstruction of aqueous outflow.

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MESECTODERMAL LEIOMYOSARCOMA OF THE ANTRUM AND ORBIT

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Secondary orbital leiomyosarcoma spreading from the paranasal sinuses has not been previously reported. We believe the leiomyosarcoma we studied is related morphologically and embryologically to a group of intraocular tumors with the light microscopic appearance of a neurogenic tumor, but with the ultrastructural features of smooth muscle. 1-2 The distinctive origin of most of the head and neck

connective tissue from the neural crest (mesectoderm) may be responsible for the bizarre hybrid differentiations of these rare intraocular and periocular smooth muscle tumors. 1,3

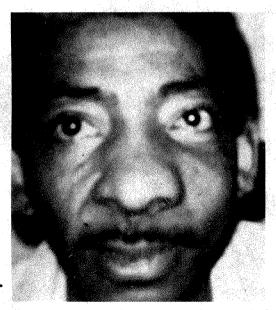
CASE REPORT

Clinical history—A 39-year-old black man was seen by the Ophthalmology Service at the Harlem Hospital Center in December 1976 for decreased vision and protrusion of the left eye of several weeks' duration. The proptosis developed 23 months after a malignant tumor of the left antrum had been diagnosed as "a sarcoma—probably a rhabdomyosarcoma." Past medical history was contributory only for hypertension and anemia.

The patient had been admitted to the hospital in December 1974, after a three-month history of sero-sanguineous discharge from the left nostril and swelling of the left cheek (Fig. 1, left). The swelling involved the left maxillary and zygomatic areas. Bloody discharge from the left nostril was purulent; the lateral nasal wall was pushed medially. An ulcerated mass was located posteroinferior to the left middle turbinate. Results of ophthalmologic exami-

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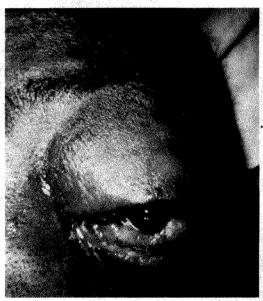
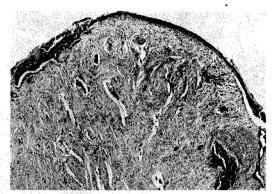


Fig. 1 (Jakobiec and associates). Left, Patient had swelling of his left cheek and nasal discharge. Right, Two years later the patient developed significant proptosis from direct spread of the tumor into the left orbit.

nation were unremarkable. Tomograms of the paranasal sinuses revealed a large mass destroying the left posteromedial wall of the maxillary antrum, the floor and medial wall of the orbit, and the lesser ing of the sphenoid. The pterygopalatine fossa and the misopharynx were also involved. A transnasal biops specimen of a maxillary sinus tumor was interpreted as a sarcoma. The tumor was considered too extensive for resection. Four months later the patient had no new complaints or signs of further spread of the tumor. He was lost to follow-up from July 1975 through January 1976, when reexamination revealed the tumor had spread. Another biopsy specimen was obtained and diagnosed as a rhabdomyosarcoma. Chemotherapy with cyclophosphamide (Cytoxan), dactinomycin (Actinomycin D). vincristine, and methotrexate was begun. The tumor had moderately regressed before the patient was again lost to follow-up. By May 1976 the patient had lost 35 pounds; he was admitted to the hospital for a second cycle of chemotherapy. Despite further local extension of the tumor, ophthalmologic examination revealed uncorrected visual acuity of L.E.: 6/9 (20/ 30) and no evidence of proptosis or motility disorder. We observed little response to the chemotherapy. The patient was admitted in August 1976 because of a drop in the hematocrit level to 18%; transfusion brought it up to 35%. He then underwent a course of radiotherapy totalling 3,800 rads. There was some improvement by the time of discharge. In October 1976 a left carotid arteriogram demonstrated increased vascularity in the left retroorbital area without a definite tumor stain.

The emaciated patient was admitted in November 1976 for possible surgical treatment after the inadequate response to chemotherapy and radiotherapy. On examination the visual acuity was R.E.: 6/6 (20/20), and L.E.: no light perception (Fig. 1, right). Marked left proptosis had produced hyperemia, chemosis, and prolapse of the conjunctiva. Left extraocular muscle movement was severely limited in all directions; the pupil was dilated and nonreactive. The cornea displayed exposure keratitis, which obscured a view of the fundus. An exenteration of the left ethmoid and maxillary sinuses, as well as of the left orbital contents, was performed. Intraoperative findings included destruction of the entire left maxilliary antrum along with the floor, roof, and medial and lateral walls of the orbit; also, the hard palate was eroded. Nine months postoperatively the patient's condition was stable, without any documented spread of the tumor or additional functional deficits, although he had a residual tumor.

Pathologic findings—Sections of the first biopsy specimen taken from the antral mass revealed a tumor growing beneath the sinus mucosa that showed squamous metaplasia (Fig. 2, top). The tumor exhibited two morphologic aspects. One was a compact growth pattern containing sclerosed blood vessels with concentrically laminated collagen bundles (Fig. 2, top). In these areas the tumor cells formed short bundles, often manifesting palisading nuclei (Fig. 2, bottom); the background cytoplasmic



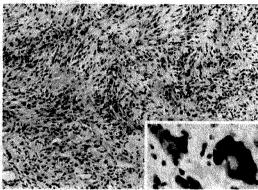


Fig. 2 (Jakobiec and associates). Top, Solid area from the first biopsy. Tumor is situated beneath the antral mucosa and displays prominent vessels with sclerotic walls. Bottom, Less vascular area showing a distinctly neurogenic appearance with palisading nuclei and short, loosely textured fasciculi. Inset, Abnormal multinucleated cells scattered throughout the tumor (hematoxylin and eosin; top, ×38; bottom, ×80; inset, ×370).

matrix was loosely textured and fibrillar, imparting a neurogenic appearance. The second prominent pattern of this biopsy material was of myxoid tissue punctuated by blood vessels (Fig. 3, left). Some of these channels displayed a cuff or a few atypical cells (Fig. 3, top right). Other areas were characterized by a pseudohemangiopericytomatous pattern of interanastomosing capillary-sized channels separated by tumor cells (Fig. 3, bottom right). Mitotic figures and abnormal nuclear forms were scattered throughout the tumor (Fig. 2, bottom inset). The trichrome stain revealed dense cytoplasmic fuchsinophilia. The reticulin stain demonstrated many wiry, spiral argyophilic fiber deposits, running parallel to the short fasciculi as well as surrounding individual tumor cells.

The second biopsy specimen showed a different appearance and represented extension of the tumor into the inferior orbit. Evenly dispersed ectatic

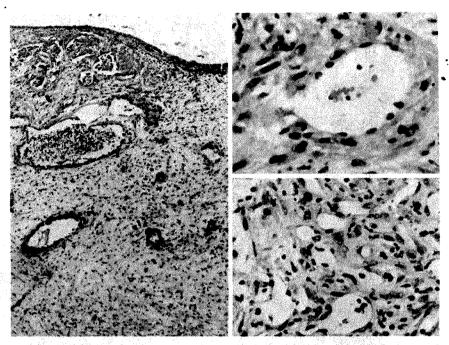


Fig. 3 (Jakobiec and associates). Left, Much of the tumor was composed of myxoid tissue. A prominent vascular pattern is preserved in this area, and the vessels have multiple layers of cells in their walls. Top right, Some of the vessel walls are composed of cuffs of tumor cells. Bottom right, A pseudohemangiopericytomatous field created by interanastomosing capillaries separated by tumor cells (hematoxylin and eosin; left, ×38; top right, ×176; bottom right, ×110).

capillary channels were separated by a population of loosely arranged tumor cells with elongated cytoplasm and many fibrillary processes (Fig. 4, top). Multinucleated giant cells and atypical bizarre tumor cells were scattered throughout the tumor. Many of the tumor cells contained cigar-shaped nuclei (Fig. 4, inset). The reticulin stain revealed numerous elongated, wavy fibers (Fig. 5); these were more loosely distributed than in the first biopsy specimen because of the more open fibrillary background. Individual cells often appeared completely surrounded by reticulin fibers. Masson's trichrome stain revealed longitudinal cytoplasmic fibrils within the tumor cells (Fig. 5, inset).

Using procedures detailed elsewhere,⁴ we performed electron microscopy on fresh tissue obtained at the time of the exenteration (Fig. 6). The tumor cells had moderate amounts of rough-surfaced endoplasmic reticulum in the perinuclear zone. Cytoplasmic filaments with fusiform densities were aggregated in the periphery of the perikaryon (Fig. 6), but they dominated the tumor cell processes (Fig. 6, inset). Interrupted basement membrane formation was seen occasionally around the tumor cells; pinocytotic vesicles were numerous. Ultrastructural studies performed on deparaffinized tissue⁵ from the

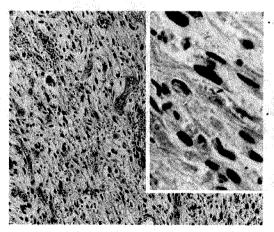


Fig. 4 (Jakobiec and associates). The second biopsy was composed entirely of loosely textured, fibrillated tumor cells with scattered tumor giant cells. Inset, The fibrillar character of the tumor cells is apparent. Many of the cells have cigar-shaped nuclei (hematoxylin and eosin; ×94; inset, ×375).

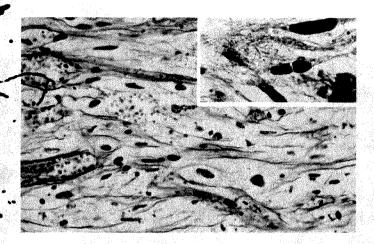


Fig. 5 (Jakobiec and associates). Numerous reticulin fibers surround individual tumor cells. Inset, Longitudinal cytoplasmic fibrils (Laidlaw-reticulin, ×192; inset, Masson's trichrome, ×600).

second biopsy specimen disclosed similar, but less
 well-preserved, morphologic features.

DISCUSSION

After several surgical pathologists reviewed the microscopic slides prepared from the first biopsy specimen, the most frequent diagnosis was a malignant. Schwann cell tumor. The myxoid areas and the palisading of nuclei in the more fibrillar and fascicular zones suggested this diagnosis. Cytoplasmic trichrome positivity and numerous tumor giant cells in the second biopsy specimen led to the diagnosis of a rhabdomyosarcoma. The patient then received radiation and chemotherapy.

Several light microscopic features should have militated against the diagnosis of either a malignant Schwann's cell tumor or a rhabdomyosarcoma. The prominent vascular pattern (pseudohemangiopericytomatous in areas) is not typical of either of these tumors, but may be well developed in smooth muscle tumors. 6-8 The atypical cells in some of the blood vessel walls furnish additional proof of the cytogenetic relationship of these channels to the tumor; the vessels in Schwann cell tumors are hyalinized rather than cellular or thickly collagenized. The heavy reticulin fiber deposition can

be seen in Schwann cell and smooth muscle tumors, but is not characteristic of rhabdomyosarcomas. The presence of numerous, classic cigar-shaped nuclei should have suggested the diagnosis of a smooth muscle tumor. 6-8

The electron microscopic studies performed on deparaffined biopsy material and on fresh tissue obtained later and fixed in glutaraldehyde confirmed the smooth muscle origin of the tumor. All of the tumor cells in both samples contained variable numbers of thin cytoplasmic filaments with fusiform densities, which were concentrated mostly in the cytoplasmic processes. Rhabdomyosarcoma cells contain thick and thin cytoplasmic filaments with some sarcomeric organization but without fusiform densities,9 while all kinds of cytoplasmic filaments are sparse in peripheral neurogenic tumors. 10,11 In the present tumor, basement membrane formation was spotty, as expected in a poorly differentiated, malignant tumor with rapidly dividing cells. Also, the rough-surfaced endoplasmic reticulum was better developed than in end-stage benign smooth muscle cells.6 but was consistent with what has been described in malignant smooth muscle tumors elsewhere in the body.12 Spindle cell carcinoma cells may display thicker

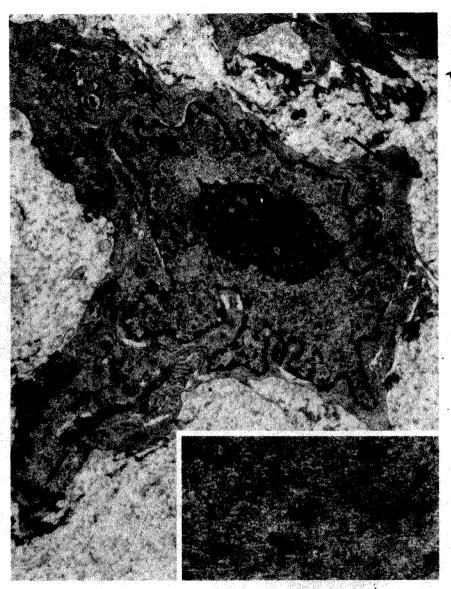


Fig. 6 (Jakobiec and associates). Top, Tumor cell with a prominent nucleolus (Nu) demonstrates cytoplasmic filaments with fusiform densities (arrows) concentrated in the periphery of the perikaryon. Inset, Tumor cell processes are occupied by cytoplasmic filaments with fusiform densities (F) to the exclusion of other organelles (electron microscopy, ×14,400; inset, ×52,000).

tonofilaments glued together by dense keratohyalin, rather than by fuzzy fusiform densities. The only cell type the present tumor cells could be confused with is the myofibroblast, which combines smooth muscle and fibroblastic traits and is found in granulation tissue¹³ and in some mesenchymal tumors.¹⁴ In these conditions thin filaments are present patchily in the cytoplasm and not in every cell, thus contrasting with the present tumor. Considering the light micro-

scopic appearance of this tumor, the electron microscopic findings supported its smooth muscle nature,

Nevertheless, the histologic appearance of the present tumor is unlike that of any berlign or malignant smooth muscle tumor that has been described in the soft tissues of the orbit.6.7 One of us (F.A.I.) thought the tumor bore a close morphologic resemblance to several benign intraocular smooth muscle tumors that had created diagnostic confusion in the past. particularly with respect to neurogenic tumors.1.2 Jakobiec and co-workers1 reported two benign tumors of the ciliary body, which like the present tumor, had a light microscopic neurogenic appearance because of a fibrillary background, but had the electron microscopic features of aberrant smooth muscle cells. A vascular smooth muscle tumor of the choroid² (proven electron microscopically), considered by light microscopy to be an unusual neurogenic or astrocytic tumor, probably also belongs to the same family of neoplasms; published illustrations2 of that tumor (Fig. 2, top and bottom) are identical to those in this study (Fig. 4). These tumors have been called "mesecto-.dermal leiomyomas."

The concept of mesectodermal mesenchymal tumors has not been published in ophthalmic periodicals. The term "mesectoderm" refers to the massive contributions of the neural crest to the connective tissues of the orbit, resulting from the absence of mesodermal somites and the limited amounts of paraxial mesoderm in these regions. Results of experimental embryologic studies on animals have demonstrated that cephalic neural crest cells contribute to the smooth muscle of the ciliary body, the pericytes of the choroid, the vascular smooth muscle cells and pericytes of the orbit, and nearly all of the head and neck mesenchyme. 1,3,15 These neural crest mesenchymal cells may rarely display hybrid tumor differentiations combining both neural and mesenchymal cellular features. We believe the present tumor arose from vascular smooth muscle cells or pericytes of the antrum because of its prominent vascularity. We suggest that its atypical appearance results from the probable neural crest origin of these elements.

Fu and Perzin⁸ studied two leiomyomas and six leiomyosarcomas among 256 nonepithelial tumors of the paranasal sinuses and nasal cavity. Among their six leiomyosarcomas, one produced proptosis; but four other tumors eventually caused orbital complications resulting either from tumor spread or surgery. Both the leiomyomas were located in the nasal cavity and did not produce ocular symptoms. These authors commented on the prominent vascularity of their tumors and speculated they arose from vessel associated cells. None of their tumors displayed the myxoid and neurogenic appearance of our case. Their patients with leiomyosarcomas fared poorly because of the invasive nature of the tumors and the reluctance of the surgeons to perform the requisite radical surgery. As with our patient, radiation therapy and chemotherapy were ineffective. Thus, because of multiple recurrences and a high fatality rate, a guarded prognosis is required.

SUMMARY

A 39-year-old black man developed a left antral leiomyosarcoma that subsequently extended into the ipsilateral orbit. On light microscopic appearance of the antral tumor the presence of a distinctive myxoid and fibrillar cytoplasmic background suggested the diagnosis of a malignant Schwann's cell tumor. A second biopsy of the orbital extension displayed the same fibrillar character, but the emergence of tumor giant cells with cytoplasmic trichrome positivity also raised the possibility of a rhabdomyosarcoma. Electron microscopy demon-

strated the smooth muscle derivation of the tumor, which probably originated from vascular smooth muscle elements. The atypical neural appearance of this myogenous tumor may have been caused by the extensive neural crest contribution to the cephalic connective tissues (mesectoderm). Radiation therapy and chemotherapy were ineffective.

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ORBITAL DERMOID DIAGNOSED BY COMPUTED TOMOGRAPHIC SCANNING

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The diagnosis of orbital dermoids and epidermoids by computed tomography has been previously reported.¹⁻³ On the computed tomographic orbital scan these lesions have a characteristic appearance; they often lie in the lacrimal fossa and displace the lateral rectus muscle medially. Hilal and Trokel¹ stated:

Dermoids have a density equivalent to surrounding muscle and, unlike what is expected from their composition, do not have negative Hounsfield values.

Recently, two articles demonstrated negative Hounsfield values (x-ray attenuation lower than that of water) in these lesions, thus verifying their fatty content.^{2,3} We report the case of an orbital dermoid in which the diagnosis was made preoperatively on the basis of negative Hounsfield values shown by computed tomography.

CASE REPORT

A 20-year-old man had a four-month history of an enlarging mass at the right superior orbital margin.

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Old photographs showed a right blepharoptosis had been present for approximately four years. Uncorrected visual acuity in each eye was 6/6 (20/20). A 15 × 8-mm, firm, discrete, painless mass protruded from under the lateral portion of the superior orbital rim of the right eye and extended into the orbit in the area of the lacrimal gland fossa. The mass came forward with retropulsion of the globe, but resistance to retropulsion was equal in each eye. Hertel exophthalmometer readings with a 95-mm base, were 11 mm in the right eye and 9.5 mm in the left eye. Levator palpebral superioris muscle function measured 14 mm in each eye. No abnormality of the palpebral portion of the lacrimal gland was noted. There were no preauricular or submandibular nodes. Motility was normal. Results of both the remaining ocular examination and the general physical examination were normal.

Conventional skull radiographs did not show either bony erosion or lucency in the lateral orbit to indicate a lipoma. Instead, an oblique view demonstrated a soft tissue mass measuring 1×1.5 cm in the area of the lacrimal gland. B-scan ultrasonography demonstrated a mass lesion in the region of the lacrimal fossa, outside the muscle cone, approximately 1.5 to 2.0 cm in the anteroposterior dimension. A computed tomographic scan demonstrated a cystic space-occupying lesion, lateral and superior to the right globe, displacing the lateral rectus muscle medially (Fig. 1, top left and right). The attenuation coefficient was -117 Hounsfield units (1,000 scale, in the disappearance mode) (Fig. 1, bottom).

A lateral orbitotomy revealed a cystic, encapsulated mass within the periorbita, above the fascia of the levator muscle, attached to the orbital wall at the sphenofrontozygomatic sutural junction. During the excision the mass was inadvertently ruptured, causing the extrusion of hair with cheesy and oily material. The completely excised mass was 18 mm in its greatest dimension. Histopathologic diagnosis was dermoid cyst of the orbit (Fig. 2). The postoperative course was uneventful.

DISCUSSION

The application of computed tomographic scanning to the diagnosis of or-





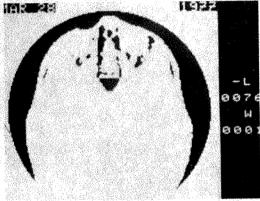


Fig. 1 (Blei and associates). Top left, Computed tomographic scan through superior orbital area showing dermoid cyst on the right. Top right, CT scan through midorbit. Bottom, Computed tomographic scan in measure mode demonstrating the negative Hounsfield number to the right of the image.

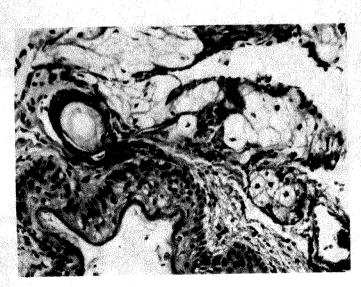


Fig. 2 (Blei and associates). Photomicrograph of cyst wall showing stratified squamous epithelium and adnexal structures.

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bital disease has been well documented. 4-22 The greatest value of the computed tomographic scan is in demonstrating the anatomy of the orbit and especially in measuring the densities of the various tissues. In our case the attenuation values in the negative range led us to the diganosis of a fat-containing tumor, most likely a dermoid cyst.

Dermoids and epidermoids are relatively common orbital lesions, totaling about 4% of all the causes of exophthalmos.²³ Since fat is the body tissue typically showing negative Hounsfield numbers. the differential diagnosis is limited. Duke-Elder²⁴ referred to a group of dermoids as "oil cysts"; these are filled with an oily liquid similar to the dermoid cyst in this case. Other less common fatcontaining tumors are lipomas and liposarcomas. Less common entities to be included in a differential diagnosis are hamartomas, eosinophilic granulomas, and early Hand-Schüller-Christian disease.23 The lesion's position in the orbit. its cystic appearance, and well-defined margins made a dermoid cyst the most likely diagnosis.

Computed tomographic scanning of the orbit could have been used alone in the preoperative examination of this patient. Other diagnostic studies of the orbit, such as cerebral angiography, orbital venography, and orbitography, would not have added significant diagnostic information.

SUMMARY

In a 20-year-old man, computed tomography revealed that an orbital mass in the region of the lacrimal gland fossa had low density attenuation values (below water), indicating a fat-containing tumor. At surgery, we found a dermoid cyst of the oil cyst type.

ACKNOWLEDGMENT

Histopathologic diagnosis was also made by the Ophthalmic Pathology Divison of the Armed Forces Institute of Pathology.

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OPHTHALMIC MINIATURE

"..., and an ivory-handled knife with a very delicate inflexible blade marked Weiss & Co., London.

"This is a very singular knife," said Holmes, lifting it up and examining it minutely. "I presume, as I see blood-stains upon it, that it is the one which was found in the dead man's grasp. Watson, this knife is surely in your line."

"It is what we call a cataract knife," said I.

"I thought so. A very delicate blade devised for very delicate work. A strange thing for a man to carry with him upon a rough expedition, especially as it would not shut in his pocket."

Sir Arthur Conan Doyle, *The Annotated Sherlock Holmes*, W. S. Baring-Gould, editor. New York, Potter, 1967, p. 271

DERMIS-FAT GRAFT AS A MOVABLE IMPLANT WITHIN THE MUSCLE CONE

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Exposure of an orbital implant is a complication that may follow routine enucleation or evisceration. Various methods of repair such as scleral grafts, fascia lata, temporal fascia, and autogenous dermis have been used to prevent extrusion. Persistent recurrent exposure and repeated surgical failures usually result in contracture of the socket and eventual extrusion of the implant from within the muscle cone.

During the past two years, we have used an autogenous dermis-fat graft to replace the exposed implant. Persistent and recurrent exposure with secondary infection around the implant requires a permanent single procedure. We believe the final result is accomplished best by an autogenous dermis-fat graft.

The graft is transplanted to the orbit from the area of the hip. This technique ameliorates the cosmetically disturbing effects of enophthalmos and the deep supratarsal sulcus that often accompany the migrating or extruding implant.

MATERIAL AND METHODS

We performed palpation of the socket to confirm the presence and location of the implant (Fig. 1, A). A horizontal conjunctival incision was made along the central portion overlying the implant (Fig. 1, C). The dissection was carried through Tenon's capsule to expose and remove the implant (Fig. 1, D). If the implant had migrated, an attempt was made to

locate the central cavity of Tenon's capsule, which would receive the dermisfat graft. Once identified, the cavity was incised in several places through Tenon's capsule to provoke more rapid vascularization of the donor fat graft (Fig. 2, E).

Upon removal of the graft from the lateral aspect of the thigh, we transported it gently (Fig. 2, F) to the recipient site. The dermis-fat graft was sutured to the anterior edge of the cavity comprising conjunctiva and Tenon's capsule (Fig. 2, G). We placed the initial 4-0 chromic sutures through the stumps of the rectus muscles at the 12, 3, 6, and 9 o'clock positions. Engagement of the rectus muscle in addition to conjunctive and Tenon's capsule enhanced the motility of the socket. Additional sutures were added in the appropriate quadrants. A doughnut conformer or a standard conformer was placed in the socket at the end of the procedure (Fig. 2, H). A prosthesis may be fitted after four weeks.

The dermis-fat graft came from the lateral aspect of the thigh in the vicinity of the buttock (Fig. 3, A). This area has a high concentration of subdermal fat. With a dermatome, we removed 8 to 10 cm of epidermis, thus leaving the base of the epidermis at the end of the dermatome intact. We cut the cylindrical dermis-fat graft to the desired size with a Bard-Parker knife to a depth of about 4 cm (Fig. 3, C).

We closed the donor site with a subdermal 4-0 chromic suture (Fig. 3, E). The dermis was closed in a similar fashion, and the epidermis, still undisturbed at its base, was sutured with interrupted 5-0 silk to its original position (Fig. 3, F). A telfa dressing was applied to the graft site.

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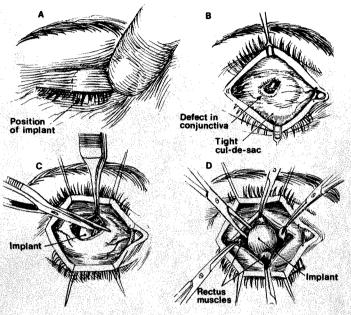


Fig. 1 (Smith and Petrelli). Palpation of the socket confirms the location of the migrating implant (A), and the opened eyelids reveal the extruding implant (B). After incising the conjunctiva (C), the implant is removed, and the rectus muscle stumps are isolated (D).

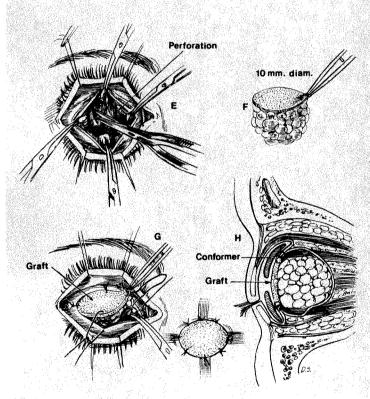


Fig. 2 (Smith and Petrelli). Host bed incisions in several places (E)-promote vascularization of the donor graft (F). The composite graft is sutured to the adjacent conjunctiva and, if possible, to the rectus muscle stumps (G). A doughnut conformer is placed in the socket (H, lateral view).

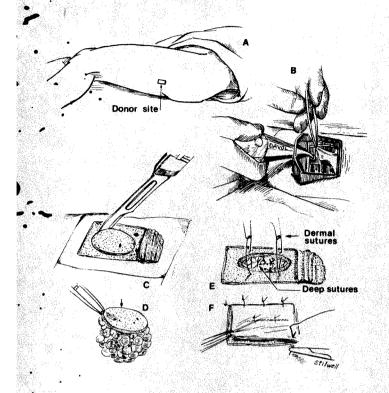


Fig. 3 (Smith and Petrelli). The dermis-fat graft is obtained from the lateral aspect of the thigh (A). The dermatome removes the epidermal layer left intact at one end (B), and the dermis-fat graft is fashioned to the desired size (C). After removal, the defect is closed with 4-0 chromic suture (E), and the flap of epidermis is closed with 5-0 silk (F).

CASE REPORT

À 31-year-old woman was referred to us in March 1977 because of an extruding implant in her right orbit. Her right eye had been removed in May 1976, after an automobile accident, and a wire mesh implant had been placed. In July 1976 the implant became exposed and the patient underwent a procedure to cover the implant. In January 1977, the implant was again exposed, but in a different location, and began producing chronic drainage. The prosthesis could not be retained.

Examination revealed an exposed orbital implant in the lateral conjunctival area of the right eye, with a draining nonpurulent fistula and socket contracture (Fig. 4). A hypertrophic scar was adjacent to the medial canthus. The patient received oral ampicillin before surgery. On April 21, 1977, the wire mesh implant was removed and a dermis-fat graft was implanted. Ten days after surgery, the conformer was in place, and the graft showed evidence of conjunctival epithelialization.

Eight weeks after surgery, the dermis-fat graft was covered with conjunctival epithelium (Fig. 5). A prosthesis was fitted successfully (Fig. 6). There has been no evidence of infection or continued drainage in the right orbit.

Of the five patients treated thus far, two had

recurrent extruding exposed implants. Three patients had migrating implants outside the muscle cone. No postoperative infections or fat graft resorptions have occurred.

DISCUSSION

Free fat grafts have been used with variable success since the early reports of Neuber³ and Lexer⁴ at the turn of the century. Rapid reabsorption of the fat graft has resulted in the abandonment of the technique. More recently, composite dermis-fat grafts have been used to build up soft tissue defects of the face, arms, legs, and breasts.^{5,7} The composite graft is more resistant to severe reabsorption and becomes more extensively vascularized.⁶ Early vascularization of the graft increases the population of fat cells capable of surviving. Watson⁷ believes the dermis has a vasoinductive effect which pro-





Fig. 4 (Smith and Petrelli). A 31-year-old woman with an extruding ocular implant in her right orbit and draining fistula near the lateral canthus.



Fig. 5 (Smith and Petrelli). Eight weeks after surgery, dermis-fat graft is covered with migrating conjunctival epithelium.



Fig. 6 (Smith and Petrelli). Twelve weeks after surgery, the patient has been fitted with a prosthesis.

motes the early beneficial vascularization of the composite graft.

Few articles pertain to the use of dermis-fat grafts in enophthalmos. Hawtof⁸ used dermis-fat strips to build up the supratarsal deformities, after traumatic enophthalmos, in the seeing eye.

In the case of migrating and extruding implants, the use of dermis-fat grafts is an alternative to secondary synthetic implants. The problems of epidermal cysts and fat graft absorption have been cited as reasons for the failure of grafts. The first problem has been solved by eliminating the epidermal elements from the dermis-fat graft. Bothersome epidermal cysts are consequently eliminated.

The second problem, fat resorption, has been reduced by the use of the composite graft. Peer⁹ reported a 45% resorption in autogenous fat grafts. Thompson¹⁰ used a subcutaneous dermis graft and reported only a 20% reduction. The prevention of bulk loss is related to good aseptic technique, gentle, expeditious handling of the composite graft, and a good vascularity of the host bed.

SUMMARY

A 31-year-old woman had an extruding ocular implant in her right orbit. After surgically removing the implant, we transferred an autogenous dermis-fat graft from the hip region to the freshly prepared socket. The graft was sutured to the conjunctiva, and a conformer wasplaced in the socket. Eight weeks postoperatively, the dermis-fat graft was covered with conjunctival epithelium and a prosthesis was fitted successfully. No evidence of infection has occurred. This technique of using composite dermis-fat grafts in enophthalmos avoids recurrent extrusions and corrects the cosmetic problems produced by migrating or extruding implants.



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OPHTHALMIC MINIATURE

The two men turned, and Lord Mayfield uttered a surprised ejaculation.

"Hallo! See that?"

"See what?" asked Sir George.

"Thought I saw someone slip across the terrace from my study window."

"Nonsense, old boy, I didn't see anything."

"Well I did-or thought I did."

"Your eyes are playing tricks on you. I was looking straight down the terrace, and I'd have seen anything there was to be seen. There's precious little I don't see—even if I do have to hold a newspaper at arm's length.

Lord Mayfield chuckled.

"I can put one over on you there, George, I read easily without glasses.'

"But you can't always distinguish the fellow on the other side of the house. Or is that eyeglass of yours sheer intimidation?"

> Agatha Christie, The Incredible Theft. Penguin Books, 1961

CHOROIDAL MELANOMA CLINICALLY SIMULATING A RETINAL ANGIOMA

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> Philadelphia, Pennsylvania AND PIERRE GUIBOR, M.D.

New York, New York

Malignant melanomas of the choroid may produce a number of clinical and histologic changes in the adjacent structures. Disruption of the retinal pigment epithelium has been associated with drusen, subretinal fluid, and accumulation of lipofuscin pigment.^{1–3} Alterations in the sensory retina may lead to cystic spaces in the outer plexiform layer and occasional retinal hemorrhages.⁴ Despite these changes in the retinal pigment epithelium and sensory retina, the retinal vessels are seldom altered over choroidal melanomas and are usually of normal caliber and distribution.

We describe herein a case of a nonpigmented fundus tumor that had a dilated tortuous retinal vessel draining the mass, similar to that seen in retinal angiomatosis. Despite the ophthalmoscopic, fluorescein angiographic, and ultrasonographic appearance of angioma, a positive radioactive phosphorus (³²P) uptake test indicated the lesion was malignant. The enucleated globe revealed an amelanotic melanoma with a large vein draining from the tumor into the retinal circulation.

From the Oncology Unit, Retina Service, Wills Eye Hospital Philadelphia (Drs. Shields and Joffe), and the Oculoplastic Service, New York Medical College, New York (Dr. Guibor). Presented at the Armed Forces Institute of Pathology Alumni Association Meeting, Washington, D.C., June 4, 1977. This study was supported in part by the Retina Research and Development Foundation, Philadelphia, and the Lions Club of Pennsylvania.

Reprint requests to Jerry A. Shields, M.D., Oncology Unit, Retina Service, Wills Eye Hospital, 1601 Spring Garden St., Philadelphia, PA 19130.

CASE REPORT

A 35-year-old white man had no ocular problems until three months before admission when he experienced an abrupt onset of "light flashes" in the right eye. His primary ophthalmologist examined him and found no abnormalities. The symptoms persisted and, five days before admission, one of us (P.G.) saw him and found a solid retinal detachment in his right eye. The patient was referred for further evaluation.

The patient was apparently healthy and had not systemic complaints. Medical history was noncontributory and there was no family history of ocular or central nervous system disease. Results of physical examination were normal.

The patient's uncorrected visual acuity was 6/6 (20/20) in both eyes; intraocular pressures were R.E.: 14 mm Hg, and L.E.: 9 mm Hg, by applanation. The fundus of the left eye and results of external and slit-lamp examinations were normal. The pertinent findings were confined to the right fundus.

Myelinated nerve fibers were present on the lower portion of the optic disk. The superotemporal retinal vein was normal as it left the disk margin, but at the first bifurcation its superior branch abruptly became dilated and tortuous. Superiorly, the vein transversed the surface of an amelanotic fundus tumor and then dipped into the substance of the tumor (Fig. 1). Ophthalmoscopically the mass appeared to involve the sensory retina. Subretinal fluid extended from the temporal margin of the mass along the ora serrata to become bullous inferiorly. Deep retinal hemorrhages were scattered along the inferior margin of the tumor (Fig. 1).

We performed several ancillary tests to make an accurate diagnosis. Fluroescein angiography demonstrated several small retinal arteries passing into the superficial portions of the lesion (Fig. 2, left). The tumor began to show mottled fluorescence during the arterial phase and progressive staining. The larger tortuous vein drained from the tumor substance into the retina and back toward the optic disk. (Fig. 2, right). Late angiograms showed marked fluorescence of the tumor and the overlying vitreous.

B-scan ultrasonography demonstrated a mass superiorly which showed moderate internal reflectivity without choroidal excavation or orbital shadowing (Fig. 3). The serous retinal detachment was demonstrated inferiorly.

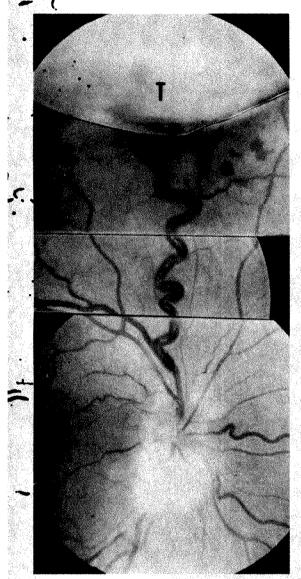


Fig. 1 (Shields, Joffe, and Guibor). Composite fundus photograph showing dilated tortuous vein draining from superior amelanotic tumor (T) toward the optic disk. The myelinated nerve fibers at the inferior margin of the disk are probably incidental. Note the intraretinal hemorrhages along the inferior border of the tumor.

A noncutting ³²P uptake test was performed by using the solid state semiconductor detector unit. Results were: 37% uptake at one hour, 81% uptake at 24 hours, and 97% uptake at 48 hours. This is considered a positive test.⁵

Results of a complete blood survey including liver enzymes, chest roentgenograms, and liver scan were normal. On the basis of ancillary diagnostic tests and the negative systemic survey, enucleation was performed without complication.

RESULTS

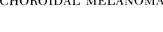
Grossly, the globe was of normal size and the anterior segment was unremarkable. The eye was sectioned vertically. A large, irregular, amelanotic mass appeared in the choroid and ciliary body superiorly. The dilated retinal vein passed from the superficial portion of the tumor toward the optic disk (Fig. 4). There was a serous retinal detachment inferiorly.

Histologic examination revealed an amelanotic choroidal tumor, which had extensively infiltrated the overlying retina. Sections of the retina near the optic disk showed the retinal vessel occupying almost the full thickness of the retina (Fig. 5). The vessel entered the portion of the retina infiltrated by the tumor (Fig. 6), and then dipped abruptly into the choroidal portion of the mass (Figs. 7 and 8). The tumor cells were a combination of spindle B and small epithelioid cells (Fig. 9). Lymphocytes were scattered throughout the tumor, particularly along the blood vessels. The myelinated nerve fibers were in a position inferior to the optic disk and were confirmed with special stains.

The final diagnoses were: (1) malignant melanoma of the choroid and ciliary body, mixed cell type, with a choroidoretinal anastomosis; (2) retinal invasion by melanoma; and (3) myelinated nerve fibers.

DISCUSSION

In this case, a nonpigmented mass involving the retina, associated with a dilated tortuous retinal vessel, clinically suggested a retinal angioma. In contrast to retinal angiomas, however, the retinal arteries passing toward the tumor were not dilated or tortuous and may not have contributed to its blood supply. Addition-



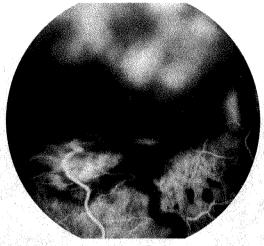


Fig. 2 (Shields, Joffe, and Guibor). Left, Early fluorescein angiogram taken during the arterial phase. Note the relatively normal retinal arteries passing toward the tumor, which shows early mot-tled fluorescence. Right, Angiogram during the arteriovenous phase. Note the increased fluorescence of the tumor and the dilated tortuous retinal vein draining the mass.

ally, fluorescein angiography could not substantiate the presence of a retinal blood supply to the tumor. Dilated retinal vessels may also be seen with other retinal tumors, such as retinoblastoma. Although Reese⁶ has shown that choroidal melanomas may invade the retina and

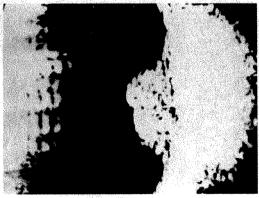
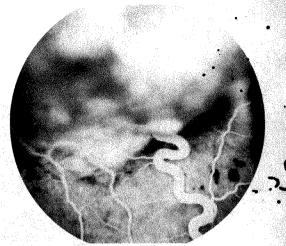


Fig. 3 (Shields, Joffe, and Guibor). Contact B-scan ultrasonogram showing intraocular tumor with moderate internal reflectivity without choroidal excavation.



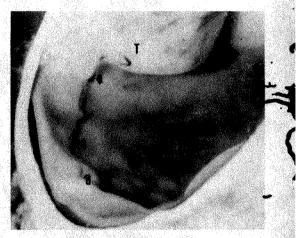


Fig 4 (Shields, Joffe, and Guibor). Section of gross globe showing amelanotic tumor (T) and large dilated retinal vein (arrows) passing from tumor to optic disk (d).



Fig. 5 (Shields, Joffe, and Guibor). Photomicrograph of retina near the optic disk showing several sections through the tortuous retinal vessel. Note that the vessel extends into the photoreceptor layer. (hematoxylin and eosin, × 40)



Fig. 6 (Shields, Joffe, and Guibor). Cross section of retina near apex of tumor showing the large vessel (arrows) passing through the retina, which has been completely replaced by melanoma cells. (hematoxylin and eosin, ×40)



Fig. 7 (Shields, Joffe, and Guibor). This section, near the apex of the mass, shows segments of the same vessel within both retinal (black arrow) and choroidal (white arrow) portions of the tumor (hematoxylin and eosin, \times 100)

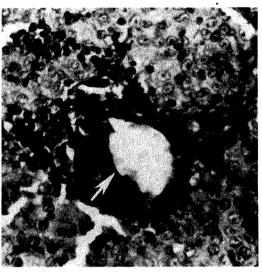


Fig. 8 (Shields, Joffe, and Guibor). Photomicrograph of tumor in its outer portion near the sclera, showing a section of the large vessel (arrow) which represents a continuation of the same vessel seen in Figure 7. (hematoxylin and $\cos in$, \times 150)

communicate with its blood supply, such an occurrence is rare.

Because the treatment of choroidal melanomas⁷ differs from the treatment of

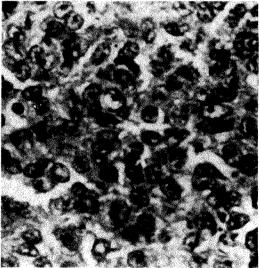


Fig. 9 (Shields, Joffe, and Guibor). High-power photomicrograph of tumor cells showing combination of spindle B and small epithelioid melanoma cells. (hematoxylin and eosin, × 250)

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retinal angiomas8, an accurate diagnosis is • important. The 32P test contributed to the correct differentiation in this case. 9.10 Fluorescein angiography was of little benefit because the large vessel and extensive late staining of the vitreous are features more compatible with angioma. B-scan ultrasonography is reportedly helpful in differentiating choroidal melanomas from lesions that simulate melanomas.11,12 In this case, we were unable to make the differentiation with B-scan. Modern methods of A-scan ultrasonography, particularly in expert hands, might have made the differentiation in our case. 13,14

The ³²P test is considered highly accurate in differentiating benign from malignant intraocular tumors. ^{5,15–17} The test was positive in this case, suggesting the lesion was malignant. Although most benign lesions give negative ³²P results, the test has not been used specifically for retinal angiomas and the incidence of false-positive results in such tumors is unknown.

The dilated retinal vessel in this case apparently occurred secondary to retinal invasion by the choroidal tumor. The precise mechanism of the choroidoretinal anastomosis, however, is uncertain.

SUMMARY

An amelanotic fundus lesion in a 35-year-old man was associated with a dilated retinal vessel, thus suggesting the diagnosis of retinal angioma. Fluorescein angiography and B-scan ultrasonography were not diagnostic, but a radioactive phosphorus uptake test suggested the lesion was malignant. The enucleated globe showed a malignant choroidal melanoma drained by a large retinal vein.

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THE INCIDENCE OF MACULAR PUCKER AFTER RETINAL DETACHMENT SURGERY

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Spontaneous, idiopathic, and congenital occurrences of preretinal membrane formation in the macula have been described by Wise^{1,2} and Bellhorn and coworkers.³ Such formation is associated with retinal vein obstructions, diabetes, posterior uveitis, retinal detachment, and ocular trauma.¹ The histopathology consists of fibrous astrocytes, Müller's cells, and other glial cell types in the spontaneous avascular membranes.³

Tannenbaum and associates⁴ described the clinical characteristics of the vitreous and retina in 105 patients with macular pucker complicating retinal detachment surgery. Patients with loss of formed vitreous, multiple operations, macular detachments, and multiple attempts at subretinal fluid drainage were at greatest risk. The authors thought vitreous degeneration affecting the vitreoretinal interface was the most important determinant in the development of the preretinal membrane.

Hagler⁵ observed that macular pucker occurred most often in patients between 50 and 60 years old. Patients with vitreous hemorrhage, vitreous membranes, or diathermy treatment had the highest incidence.

Machemer and Laqua⁶⁻⁸ have demonstrated periretinal membranes that formed starfolds, rolled edge tears, and equatorial ridges throughout the retina in experimental detachments in the owl monkey. These changes were clinically indistinguishable from similar changes in human retinal detachments. The membranes developed from retinal pigment epithelial cell and glial cell metaplasia and hyperplasia.

Clinical studies to date^{4,5,9-11} stress the role of the vitreous collagen, hyalocytes, and vitreoretinal interface in the formation of macular pucker after detachment surgery.

We initiated this study to identify risk factors predisposing patients to macular pucker after retinal detachment surgery and, indirectly, to test the hypothesis that preretinal membrane formation in the macula is related not to vitreous alterations, but rather to retinal glial cell or pigment epithelial cell proliferation.

SUBJECTS AND METHODS

We obtained clinical information from 1,049 consecutive cases of retinal reattachment performed at the hospitals and clinics here. There were 863 primary procedures and 186 reoperations. Patients with retinal breaks surrounded by less than 1 disk diameter of subretinal fluid (205 cases) were excluded. By requiring a minimal follow-up period of six months, we eliminated 192 patients, thus reducing the final study population to 857. Seventy-four (8.6%) patients overall developed macular pucker, 63 (7.5%) patients developed a macular pucker as the only late postoperative complication after successful reattachment of the retina. An additional 11 patients developed macular pucker in association with other significant complications. We excluded them to acquire a more homogeneous population,

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thus minimizing multiple interrelating factors and keeping our risk factors more specific for macular pucker.

We prepared computer punch cards from a six-page data collection sheet developed with the assistance of the Department of Biostatistics and Preventive Medicine.

Fifteen major categories of independent variables contained a total of 129 linear and categorial variables. Each variable was cross-tabulated with the presence or absence of postoperative macular pucker.

Statistical analysis included the chisquare method for determining difference among proportions, the two by two chisquare test, Fisher's exact test, and partitioning of the overall chi-square test to determine trends in the proportions.¹²

RESULTS

The age-specific incidences of macular pucker were demonstrated (Fig. 1). The overall chi-square test for the entire population was not significant for age. Similarly, there was no significant difference in the group of patients aged 50 years and older as compared to the group younger than 50 years. Graphically, however, a linear trend after age 30 years was evident. Partitioning of the overall chi-

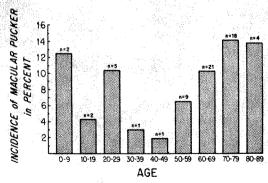


Fig. 1 (Lobes and Burton). Age-specific incidence of macular pucker among retinal detachment patients

TABLE 1
RELATIONSHIP OF BLUNT TRAUMA TO*
MACULAR PUCKER

	Macular Pucker
History of Trauma	No. Present No. (%) Absent
Present Absent	6 (10.7) 50 53 (8.8) 546 P>.05

square according to Maxwell's 12 method described the trend after age 30 years as a statistically significant linear trend with an increase in risk with advancing age (P<.05).

The study group consisted of 52.5% males and 47.5% females. The sex-specific incidence of macular pucker was 9.9% for males and 8.2% for females. The difference was not significant (P>.05).

There was no statistically significant correlation between ocular trauma and macular pucker (Table 1). The rates of macular pucker were similar for emmetropic and aphakic patients (Table 2). However, patients with greater than 4 diopters of myopia had a significantly lower rate of macular pucker (P<.05). Risk of macular pucker increased with decreasing values of preoperative visual acuity (P<.05) (Fig. 2).

On preoperative examination, uveitis was defined as 2+ flare or cells. Vitreous hemorrhage was recorded only when it impaired visualization of the fundus. Neither complication presented a statistically increased risk factor (P>.05). A history of glaucoma, the presence of a cataract, or pars plana detachment was noncontributory (Table 3).

No significant difference in the incidence of pucker among patients with posterior vitreous detachment, vitreous bands and membranes, or fibrillar changes could be demonstrated when

TABLE 2
RELATIONSHIP OF REFRACTIVE ERROR TO
MACULAR PUCKER

No.	
Present No. Refractive Error* (%) Absent	Significance
Emmetropia (-4D-+4D) 37 (11.3) 289 Myopia (-4D) 4 (4.2) 90 Aphakia (+8D-+14D) 22 (9.1) 221	Control P<.05 NS†

^{*}D denotes diopters. INS denotes not significant.

compared to patients with "normal vitreous" (P>.05 on each factor).

The category of isolated vitreoretinal adhesions represented detachment patients with no other complicating retinal disease. The 15.1% incidence of macular pucker among patients with rolled edges, starfolds, and equatorial ridges (signs of localized preretinal membrane formation) on preoperative examination (Table 4) is significantly higher (P<.05) than the 7.4%

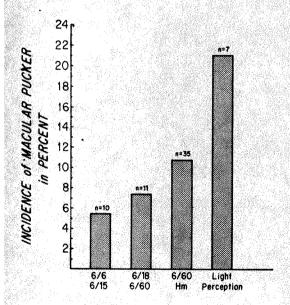


Fig. 2 (Lobes and Burton) Relationship of preoperative visual acuity to incidence of macular pucker among retinal detachment patients.

incidence among the control group. The presence of lattice degeneration or a dialysis on preoperative examination did not increase the risk of macular pucker (P>.05).

There was no statistically significant difference in the incidence of macular pucker between procedures involving episcleral exoplants (7.5%) and those involving scleral dissection and implants (9.6%). Cryopexy was used in all but six cases, so comparison between diathermy and cryopexy was impossible.

The immediate intraoperative and postoperative complications of vitreous loss, choroidal detachment, vitreous hemorrhage, and uveitis did not produce an increase in the risk of macular pucker (P>.05 in all cases). However, three of 11 patients with choroidal hemorrhage de-

TABLE 3
PREOPERATIVE COMPLICATIONS RELATED
TO MAGULAR PUCKER

	Macular	Pucker	
Complication	No. Present (%)	No. Absent	Significance
None	44 (5.9)	706	
Uveitis	1 (4.5)	21	NS
Vitreous hemorrhage	3 (4.1)	70	NS
Cataract	5 (8.3)	55	NS
Glaucoma	38 (9.5)	4	NS

TABLE 4
THE RELATIONSHIP BETWEEN TYPE OF
DETACHMENT
AND MACULAR PUCKER

	Macular	Pucker	
Type of Detachment	Present	No. Absent	Significance
Vitreoretinal	12 (7.4)	151	Control
adhesion Lattice	8 (7.1)	104	NS
Dialysis	4 (7.4)	50	NS
Preretinal membrane	27 (15.1)	151	P<.05

veloped a macular pucker and appeared to have an increased risk as indicated by the Fisher's exact test.

Additional argon laser, xenon photocoagulation, or cryotherapy during the postoperative convalescence was performed in 47 patients; three developed macular pucker. This was not significantly different from the control group (Fisher's exact test).

The incidence among 583 control patients, with one scleral buckling operation and no complication except macular pucker, was 8.2%; while among patients with more than one procedure, and no complications except macular pucker, it was 15%. This difference was statistically significant (P<.05) (Table 5).

The final visual acuity of patients developing macular pucker was significantly lower than the remainder of the patients (P<.0001). Among the 58 affected patients only 12% attained visual acuity

TABLE 5
RELATIONSHIP OF REOPERATION
TO MACULAR PUCKER

Operation	No. Present (%)	No. Absent	Significance
1	48 (8.2)	535	Control
2 or more	15 (15)	85	P<.05

of 6/15 (20/50) or better, while 48% were in the 6/18 (20/60) to 6/60 (20/200) range, and 39.6% were less than 6/60 (20/200) (Fig. 3). In five patients the final visual acuity was unknown.

DISCUSSION

While the 0 to 9- and 20- to 29-year-old age groups appeared at greater risk of developing macular pucker, attack rates were obtained from only two cases in each group. The chi-square method for determining differences among proportions indicated no actual differences for any specific age group. However, from age 30 years, there is a linear, significant increase in risk with advancing age.

Penetrating injuries and intraocular foreign body cases were excluded from consideration because direct entry of conjunctival, or other epithelial cells into the vitreous cavity could occur at the time of the injury. Blunt ocular trauma did not produce a statistically increased risk of developing macular pucker. This result is supported by dialysis patients, who had a high incidence of trauma (68%), yet failed to show an increased incidence of postoperative macular pucker.

In this study the median age of myopic detachment patients was 36 years, the greatest number occurred in the 40 to 49

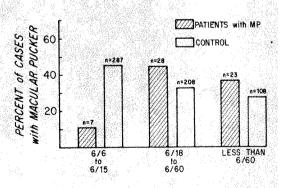


Fig. 3 (Lobes and Burton). Relationship of postoperative visual acuity to incidence of macular pucker (MP) among retinal detachment patients.

year period. Emmetropic detachment patients who had a median age of 55 years with a peak in the 60- to 69-year period. The 40- to 49-year group had a lower incidence of macular pucker than the 60-to 69-year group (linearity, P<.05) (Fig. 1). The lower incidence of macular pucker among myopic patients probably reflects the age-dependent risk factor.

Tannenbaum and associates⁴ noted an increased risk of macular pucker with total retinal detachment. This study demonstrated an increased risk with decreasing values of preoperative visual acuity and with signs of periretinal proliferation such as starfolds, rolled edges, and equatorial ridges. Perhaps these are indirect assessments of extent or duration of detachment.

In experimental models there is a progressive degeneration of the rod and cone outer, segments and advancing periretinal membrane formation with increased duration of detachment. Set Patients with detachments of longer duration would be expected to have a greater reduction in preoperative visual acuity and more advanced periretinal proliferation, of which macular pucker may be an extension. Data on duration of detachment were not available for analysis in this study.

We were unable to confirm the stated correlation between vitreous loss during scleral buckle surgery and macular pucker.⁴ Vitreous loss occurred only 23 times in our 1,049 cases, and two patients developed a macular pucker. The low incidence of this complication precludes relating it to macular pucker. None of the vitreous characteristics significantly increased the risk of developing macular pucker; this finding decreases the importance of the vitreous gel or co-existing vitreous hemorrhage as contributing factors to premacular membrane formation.

Additional postoperative xenon photocoagulation, argon laser, or cryopexy treatments failed to influence the inci-

dence of macular pucker; this challenges the hypothesis that these modalities stimulate metaplastic and proliferative responses in the macula.

We cannot satisfactorily explain the significant correlation between choroidal hemorrhage and macular pucker.

Periretinal membrane formation was the most common cause of failure in primary detachment surgery, as it was present in 65% of those undergoing reoperation, as opposed to only 28% of primary cases. We have confirmed the correlation of an increased incidence of macular pucker with more than one scleral buckling procedure. Patients requiring re-operation to achieve retinal reattachment, therefore, have significantly higher incidences of macular pucker and periretinal membrane formation. The preoperative presence of periretinal membrane formation increases the risk of macular pucker. All three factors must be closely interrelated. Macular pucker may be only a reflection of a common denominator (periretinal membrane formation), which resulted in the failed detachment surgery.

This study confirms that macular pucker is an important cause of reduced visual acuity after retinal detachment surgery and isolates several significant variables. Myopic patients have a lower risk than emmetropic patients. The incidence increases with age after age 30 years. Eyes with preoperative visual acuity worse than 6/15 (20/50) are at risk. The presence of starfolds, rolled edges on the tear, or equatorial ridges preoperatively increases the risk. Patients undergoing reoperations for failed detachment surgery have a greater risk of developing a premacular membrane.

These factors, while associated with higher incidences of macular pucker, are not causative, but predictive indicators.

We were unable to confirm previous work suggesting that degenerations of the vitreous gel, preoperative vitreous hemor-

rhage, or vitreous loss during detachment surgery produce and increase the risk of macular pucker. No significant correlation could be determined for aphakia, blunt trauma, uveitis, glaucoma, cataracts, surgical method, postoperative procedures, or refractive error.

Potentially important factors, which we did not study, include duration of detachment, drainage vs nondrainage, extent of buckle, longer follow-up, and retinal status of fellow eye.

Our data suggest macular pucker after detachment surgery is produced by a mechanism similar to that producing starfolds, equatorial ridges, and massive preretinal retraction. In the experimental detachment model, this mechanism is a periretinal membrane derived from the proliferation of retinal glial cells and pigment epithelial cells. The risk factors isolated tend to discount a causative role of the vitreous in the formation of premacular membranes.

SUMMARY

In a study of 63 cases of macular pucker among 857 patients who underwent retinal detachment repair, we isolated several statistically significant risk factors: preoperative vision of less than 6/15 (20/50); preoperative presence of rolled edges, starfolds, or equatorial ridges; re-operations; choroidal hemorrhage; and age past 30 years. We were unable to confirm the risk associated with vitreous loss, vitreous hemorrhage, and degeneration. Results

suggested that macular pucker after detachment surgery is produced by a membrane similar in origin to those causing other forms of periretinal proliferation.

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SUBRETINAL NEOVASCULARIZATION AND PAPILLEDEMA ASSOCIATED WITH PSEUDOTUMOR CEREBRI

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Subretinal or choroidal neovascularization has been previously described in a number of conditions including angioid streaks,¹ senile macular degeneration,² choroidal rupture,³ myopic retinopathy,⁴ optic nerve drusen,⁵ and histoplasmic choroiditis,⁶ as well as after argon laser photocoagulation.⁷

This report describes a case of subretinal neovascularization originating adjacent to the optic disk in a patient who had papilledema.

CASE REPORT

A·31-year-old white man was seen for routine ophthalmologic examination on June 3, 1976. He had a history of blurred vision in his right eye and recurrent frontal headaches, which had been worse for three weeks before examination. His internist had found microscopic hematuria on a routine physical examination four weeks before ophthalmological evaluation. Results of cystoscopy and an intravenous pyelogram were normal. Tetracycline therapy was begun and continued for three weeks.

Ocular examination revealed a visual acuity of 6/6 (20/20) in each eye. Trace flare and cells were in the anterior chamber of the right eye. Ocular motility, pupillary reactions, and intraocular pressures were within normal limits. Examination of the fundi with slit lamp and Goldmann contact lens revealed R.E.: 3 + elevation of the optic nerve head; and L.E.: 2 + elevation. Mild dilatation of the disk capillaries, mild peripapillary retinal edema, and retinal striae were noted. A tiny subretinal hemorrhage was seen next to the right optic disk.

On June 8, 1976, fluorescein angiography (Fig. 1) confirmed papilledema with dilatation of disk capillaries and minimal papillary fluorescein leakage. Additionally, a small area of hyperfluorescence was seen at the superior temporal disk margin. Skull roentgenograms and brain scan were negative. Two weeks later, the retinal veins were more congested and the patient was admitted to the hospital.

Neurologic examination revealed an alert, welloriented man with bilateral papilledema. Corneal sensation was equal. Motor strength, coordination, and reflexes were normal. Vital signs were within normal limits: blood pressure, 120/70; pulse 84 beats per minute; temperature 37.5°C. Results of the remaining general physical examination were normal.

Laboratory studies yielded the following results: hematocrit 40%; hemoglobin, 13.7 g/100 ml; white blood cells, 7000/mm³ with normal differential; sedimentation rate, 28 mm/hr; blood urea nitrogen, 14 mg/100 ml; blood glucose, 117 mg/100 ml; urinalysis, occasional white blood cells, no red blood cells. Results of other laboratory tests, including serum electrolytes and serum protein electrophoresis, were normal. Chest roentgenogram and electrocardiogram were also normal.

Lumbar puncture revealed an opening pressure of 370 mm H₂O, four white blood cells, no red blood cells, glucose of 63 mg/100 ml, and protein level of 20 mg/100 ml. Culture and cerebrospinal fluid fluorescent treponema antibody were negative. Electroencephalogram, echoencephalogram, and computed axial tomography were normal.

A diagnosis of pseudotumor cerebri was made, and the patient began receiving 16 mg of dexamethasone daily. His headaches gradually subsided, and the dexamethasone was tapered to 8 mg daily on the day of discharge.

Slit-lamp ophthalmoscopy was repeated on July 6, 1976; at that time the papilledema was decreasing. As the papilledema decreased, the area of juxtapapillary subretinal hemorrhage and subretinal gray membrane gradually increased. There was minimal serous detachment of the retina, and I chose to continue observation. Over the next several months, the neovascular membrane slowly extended superiorly and temporally from the disk.

On Feb. 8, 1977, fluorescein angiography (Fig. 2) confirmed a large area of subretinal neovascularization, partially pointed toward the fovea. Subretinal fluid has increased in the papillomacular bundle. Heavy argon laser coagulations were applied to the neovascular membrane.

Fluorescein angiography was repeated in three weeks (Fig. 3) and showed absence of subretinal neovascularization. Visual acuity had stabilized at R.E.: 6/12 (20/40), and L.E.: 6/6 (20/20).

DISCUSSION

In 1974, Wise, Henkind, and Alterman⁵ reported several cases of optic disk drusen and subretinal neovascularization originating next to the disk. The present case resembles those cases with optic disk drusen, except that my patient had papil-

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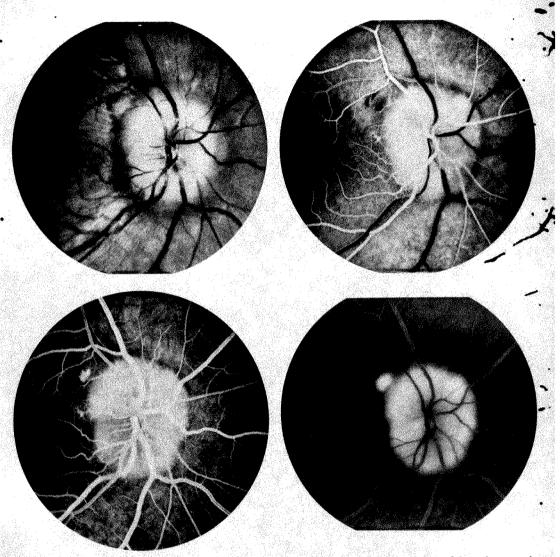


Fig. 1 (Jamison). Right optic disk. Top left, Note marked blurring of disk margins on red-free photograph. Top right, bottom left and right, Fluorescein angiogram revealed hyperemia and congestion of disk capillaries. Top right, Arterial phase. Note the small area of blocked fluorescence (subretinal blood) adjacent to the superior temporal disk margin. Bottom left, Fluorescent intensity in this spot increases in venous phase. Bottom right, Late leakage is seen.

ledema associated with pseudotumor cerebri.

At the onset, the area of peripapillary subretinal hemorrhage was tiny and could easily have been overlooked because of papilledema. As the subretinal neovascular membrane enlarged over the course of several months, it became evident that it would involve the fovea. Argon laser photocoagulation was uti-

lized to obliterate the subretinal neovascular membrane.

Argon laser photocoagulation adjacent to the disk and in the papillomacular bundle carries significant risk of nerve fiber damage even if subretinal fluid is present. The risk from treatment must be carefully weighed against the natural course of the disease.

Wise, Henkind, and Alterman⁵ specu-

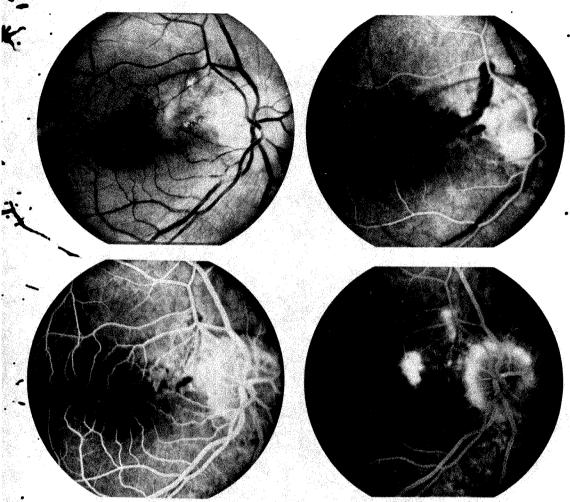


Fig. 2 (Jamison). Right optic disk and macula. Top left, Red-free photograph. Top right, bottom left and right, Note extensive grayish subretinal neovascular membrane. Top right, Fluorescein angiogram reveals blocked fluorescence in arterial phase. Bottom left, A lacy hyperfluorescence is seen in venous phase. Bottom right, Late leakage is noted.

lated that the subretinal neovascularization associated with disk drusen evolved from alteration of disk and peripapillary circulation. It would seem equally likely, however, in this case and in theirs, that mechanical distortion of peripapillary tissue could cause a break in Bruch's membrane with subsequent extension of choriocapillaris into the subretinal space.

SUMMARY

A 31-year-old man, on routine ocular examination, was found to have bilateral

papilledema. Neurologic evaluation confirmed elevated cerebrospinal pressure with no mass lesion and a diagnosis of pseudotumor cerebri was made.

A tiny subretinal hemorrhage adjacent to the right optic disk was found to be secondary to subretinal neovascularization. Over the course of several months, the papilledema resolved. However, the neovascular membrane extended further toward the fovea and was subsequently obliterated with argon laser photocoagulation.

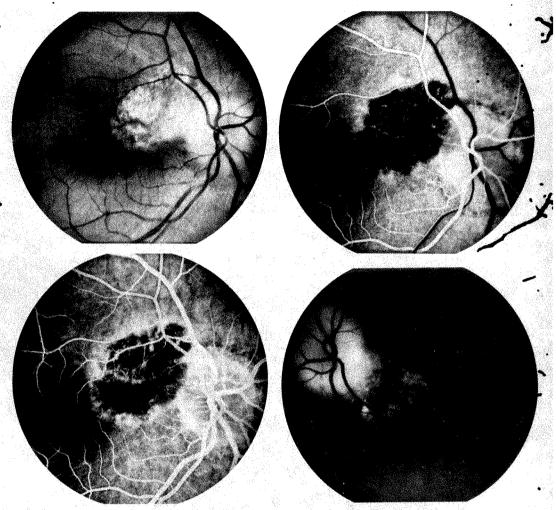


Fig. 3 (Jamison). Right optic disk and macula three weeks after argon laser photocoagulation. Top left, Red-free photograph shows extent of scarring in the papillomacular area. Top right, bottom left and right, Fluorescein angiogram. Top right, Arterial phase shows blocked fluorescence with some choroidal vascular fluorescence. Bottom left, Venous phase. Occasional window defects are noted. Bottom right, Forty minutes post injection. Fading of fluorescence is noted in the scar.

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RUBELLA RETINOPATHY AND SUBRETINAL NEOVASCULARIZATION

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Choroidal neovascularization with subsequent hemorrhage and disciform scarring is a common cause of visual loss in many diseases of the pigment epithelium, Bruch's membrane, and the choroid. Macular subretinal neovascularization with hemorrhage has been described in the following conditions: senile disciform macular degeneration 1-4; presumed histoplasmosis syndrome^{3,5}; angioid streak.3.6; myopia3.6, choroidal tumors and nevi3,6,7; vitelliform macular dystrophy8; and hereditary drusen.3 It has also · occurred in an idiopathic form for which no underlying etiology has been found^{3,9}; after traumatic choroidal rupture 3,10,11; in Sorsby's pseudo-inflammatory dystrophy12; and after photocoagulation with • the argon laser. 13,14 The following have been described as causes of neovascularization with hemorrhage around the disk: drusen, peripapillary choroiditis, presumed histoplasmosis syndrome, hyalin bodies, angioid streaks, and an idiopathic form.3 Peripheral subretinal hemor-. rhage from presumed subretinal neovascularization has also been reported. 15

This report documents the occurrence of submacular neovascularization and hemorrhage in three patients with a clinical diagnosis of congenital rubella retinopathy. Previous histopathologic studies of rubella have shown primary involvement of the pigment epithelium with normal appearing Bruch's membrane and choroid.

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METHODS

The patients were examined by indirect ophthalmoscopy, slit-lamp biomicroscope, and Goldmann contact lens. Fundus photography was performed on all patients. Electroretinography (ERG) and fluorescein angiography were performed on two patients. These techniques have been described in detail elsewhere. 16,17

CASE REPORTS

Case 1—In 1969, a 10-year-old boy had poor vision in the left eye. The age of onset could not be clearly determined. The mother had had rubella during pregnancy and the patient was congenitally deaf.

Ocular examination showed visual acuity of R.E.: 6/6 (20/20), and L.E.: 6/60 (20/200). The fundus in both eyes had diffuse mottling of the pigment epithelium typical of rubella retinopathy (Fig. 1). The left eye showed a subretinal macular hemorrhage, a characteristic consequence of subretinal neovascularization (Fig. 2). The patient was returned to the care of the referring physician. A

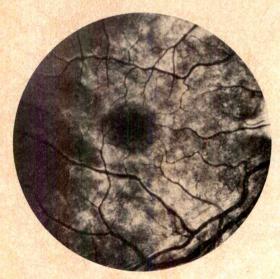


Fig. 1 (Deutman and Grizzard). Case 1. Right eye, Diffuse mottling of the pigment epithelium characteristic of rubella retinopathy.

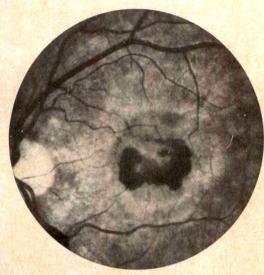


Fig. 2 (Deutman and Grizzard). Case 1. Left eye, Detachment of pigment epithelium surrounded by hemorrhage.

photograph of this patient was published previously.16

Case 2-A 7-year-old boy was seen in February 1976. He had been referred here for evaluation of a pigmentary retinopathy discovered on a routine examination. The patient was congenitally deaf and had no ocular complaints. Family history was noncontributary for deafness or ocular disease. No maternal illnesses were known to have occurred during

Visual acuity in the right eye, with +1.50 + 3.00 \times 30, was 6/12 (20/40) and in the left eye with + 1.00 + 1.00 × 95 was also 6/12 (20/40). No nystagmus was seen. The cornea and lens were normal. Fundus examination revealed a bilateral, symmetrical mottling of the pigment epithelium. The disk and vessels were normal. We made the diagnosis of rubella retinopathy and returned the patient to the referring

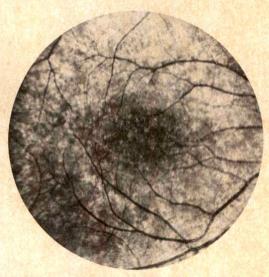
physician for further care.

The patient was again seen in January 1977 because of a sudden loss of vision in the left eye. Visual acuity in the right eye remained 6/12 (20/40) and showed the typical mottled appearance of rubella retinopathy (Fig. 3). Visual acuity in the left eye was 6/60 (20/200). The patient half developed neovascularization and hemorrh ge under the pigment epithelium and had elevation of the retina overlying the pigment epithelial detachment. A small amount of hemorrhage had entered the subretinal space (Fig. 4). Fluorescein angiography revealed neovascularization which was too near to the foveola for treatment with photocoagulation (Fig. 4, top right, bottom left and right). In April 1977, an ERG revealed normal values.

Case 3-A 14-year-old girl was seen here in June 1976. She complained of sudden loss of vision in the left eye. The patient was congenitally deaf. Family . history was noncontributary for ocular disease and deafness. There was no history of maternal illness

during gestation.

Visual acuity was R.E.: 6/6 (20/20), and L.E.: 6/30 (20/100). The cornea, anterior chamber, and lens were normal in both eyes. Diffuse mottling



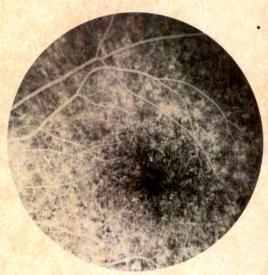


Fig. 3 (Deutman and Grizzard). Case 2. Right eye, Diffuse mottling of pigment epithelium in posterior pole, with a mottled pattern of hyperfluorence.

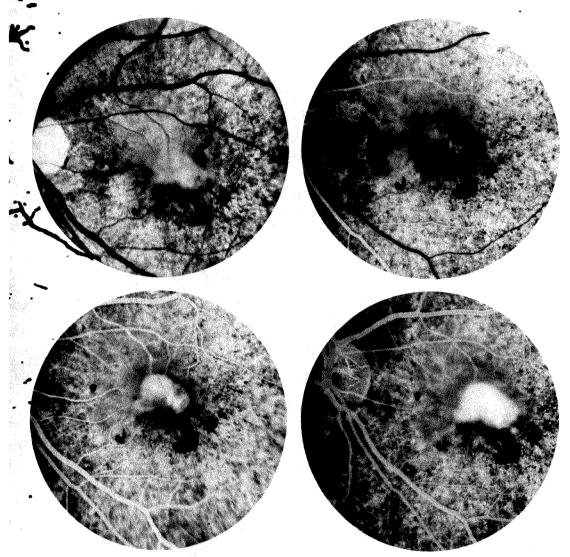


Fig. 4 (Deutman and Grizzard). Case 2. Top left, Macular hemorrhage overlying pigment epithelial detachment in left eye. Top right, Fluorescein angiogram of same eye shows early venous stage of angiogram. Delicate new vessels are visible in the foveal region. Bottom left, the accumulation of fluorescein beneath a pigment epithelial detachment later in the angiogram is clearly visible. Top right, bottom left and right, Small hemorrhages blocking fluorescence are also visible around the fovea.

of the pigment epithelium was present in each eye (Fig. 5). In the left eye an area of subretinal neovascularization with subretinal hemorrhage was seen. The neovascularization and hemorrhage were limited to the subpigment epithelial space, except at the periphery of the pigment epithelial detachment where blood had entered the subretinal space (Fig. 6). The fluorescein angiogram demonstrated a neovascular membrane and accumulation of fluorescein in the pigment epithelial detachment (Fig. 6, top right, bottom left). An ERG in April 1977 revealed mildly subnormal values for rod and cone function. We

considered the patient could be a carrier for choroideremia, but rejected the possibility of noncontributory family history and her congenital deafness. Subnormal ERGs have been reported in rubella retinopathy, though normal electroretinograms are usually found. 18,19

DISCUSSION

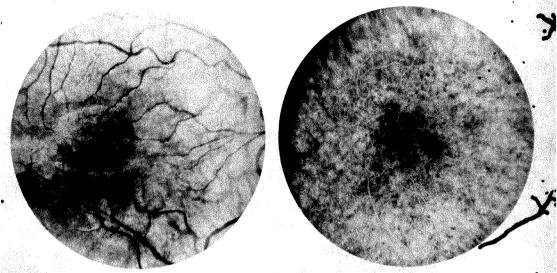
Gass¹⁻⁷ described the pathogenesis and pathology of subretinal neovascularization in senile macular degenera

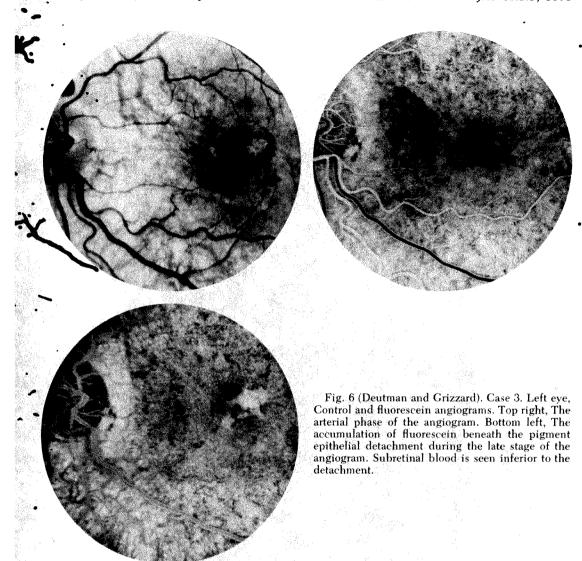
Fig. 5 (Deutman and Grizzard). Case 3. Right eye, Diffuse mottling of pigment epithelium in macula of patient with congenital rubella retinopathy.

tion, as well as the occurrence of similar choroidal neovascularization and hemorrhage in the presumed histoplasmosis syndrome, angioid streaks, myopic choroidal degeneration, choroidal tumors, an idiopathic form, and traumatic choroidal ruptures.¹⁻⁷ These observations have been confirmed.

Subretinal neovascularization has also been described as a complication of Best's vitelliform macular dystrophy and it has reportedly occurred in Sorsby's pseudoinflammatory dystrophy.^{8,12} Subretinal neovascularization has also been described as a complication after treatment with argon laser photocoagulation.^{13,14} Furthermore, we have seen late subretinal neovascularization in patients with retinochoroiditis scars caused by toxoplasmosis. Silva and Brockhurst¹⁵ have described peripheral hemorrhagic detachment of the peripheral retinal pigment epithelium.

Since Gregg originally described the clinical appearance of the rubella ocular syndrome, several clinical and pathologic studies have been made. No previously reported case of subretinal neovascularization with disciform scarring has been reported to our knowledge. In 1968 Hertzberg²⁰ reviewed 25 cases, originally described by Gregg, and noted retinopathy in 19 patients. Two patients had what he described as "choroiditis not typical of rubella." Auw²¹ noted an "abiotrophy of the Sorsby type" in one case. This may have been neovascularization with subsequent hemorrhage but was not recognized as such.

The characteristic ophthalmoscopic finding in the congenital rubella syndrome is widespread retinopathy withmottled or blotchy pigment. The macula may form pigment clumps and the foveal reflex may be lost. The ERG is usually normal but may be reduced. 18.19 Vision is usually good unless other ocular abnormalities are present. Carriers of choroideremia and ocular albinism may show a similar fundus picture. Development of the characteristic retinopathy in eyes which appeared normal when first examined has been noted to occur in the first year of life. 22



Our diagnosis of congenital rubella syndrome was based on these clinical criteria: the characteristic appearance of the fundus; congenital deafness; no family history of ocular disease or deafness; and good vision without nystagmus. One case had a normal ERG, one case had a subnormal ERG with equal cone and rod disfunction, and one case could not be tested.

Pathologic studies of rubella retinopathy have shown degeneration and depig-

mentation of the pigment epithelial cells. The absence of choroidal inflammation has been stressed. The ciliary body and iris, however, were frequently involved in inflammation with round cells, lymphocytes, plasma cells, and histiocytes. Most of these histologic specimens were obtained post mortem from neonatal deaths and therefore would not demonstrate changes which might require years to develop. ^{23,24}

The alterations present in the pigment

epithelium probably predispose rubella infants to develop choroidal neovascularization later in life. That our patients were older (9,10, and 14 years) and that one had been previously examined and found to have no neovascularization support that hypothesis.

SUMMARY

Diseases that primarily affect the pigment epithelium, Bruch's membrane, or the choroid may lead to secondary subretinal neovascularization and its sequelae of hemorrhage and scarring. We studied three cases of presumed congenital rubella retinopathy with congenital deafness, which developed unilateral subretinal neovascularization, hemorrhage, and scarring.

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HYDROCEPHALY, CONGENITAL RETINAL NONATTACHMENT, AND CONGENITAL FALCIFORM FOLD

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Thirteen patients with congenital hydrocephaly, microphthalmia, and congenital unilateral or bilateral retinal nonattachment have been described. Two cases reported by Svedbergh³ can be added to this list (Fig. 1). I previously suggested that such patients might suffer rom a nosologically distinct syndrome. 1,2 I describe herein the findings in a hydrocephalic patient with microphthalmia and congenital retinal nonattachment. The consanguinity of his parents suggests the syndrome is an autosomal recessive disorder; it can be delineated both from other hereditary traits with congenital retinal nonattachment and from retinopathy · of prematurity.

CASE REPORT

The patient was a 6-year-old boy whose parents were first cousins. The mother was 21 and the father 33 years old at the birth of the child. A younger brother is pnaffected, as is the rest of the family.

The patient's prenatal life, birth, and neonatal period were uncomplicated. The birth weight was 2,800 g. No oxygen was administered. He was breast-fed for six weeks. On changing to milk formula, diarrhea developed, and the boy failed to thrive. He was admitted to the hospital at the age of 3 months; the diarrhea remained unexplained. Normal values were found for the excretion of mucopolysaccharides and for serum aminoacids. rubella antigens or serologie indications of toxoplasmosis were disclosed. There were elevated levels of serum calcium, for which no explanation could be found. His right eye was microphthalmic, with complete retinal detachment. He had hydrocephalus, and a shunt was introduced at the age of 13 months

During infancy, scoliosis and secondary contractures of the arms and legs developed. He was unable to move and was considered blind at the age of 4 years.

The patient was seen in our clinic at the age of 6 years. He was severely retarded, hypotonic, and unable to sit up, even with support (Fig. 2). He had no language. The karyotype showed a normal 46 XY male.

Radiography showed delayed osseous development, comparable to that of a child of 3 years; a shunt was present with its cranial end in the right lateral ventricle and the eaudal tip in the right



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Fig. 1 (Warburg). Survey of patients with hydrocephaly, retinal detachment, and falciform folds.

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Fig. 2 (Warburg). Hydrocephaly and retinal detachment. The patient was 6 years old and unable to sit up. He was incontinent, hypotonic, and profoundly mentally retarded.

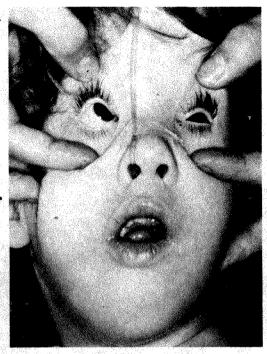


Fig. 3 (Warburg). Hydrocephaly and retinal detachment. One eye is microphthalmic, the other has a reduced corneal diameter.

atrium. The skull was only slightly larger than normal, but scaphocephalic, and there were no intracranial calcifications. Encephalography had been done at the age of 13 months. There was no communication between the lateral ventricles and the fourth ventricle, and the lateral ventricles were markedly dilated. The thickness of the brain measured 3 mm anteriorly and 1 mm posteriorly.

Radiography of all four extremities (at age 6 years) showed osteoporosis, but no fractures. Both hips were subluxated.

In the right eye (Fig. 3), the distance between the internal and external canthi was 24 mm; the horizontal corneal diameter was 7.5 mm. There was a searching nystagmus. The pupil reacted indirectly, but not directly to light. A right esotropia was present. Slit-lamp examination showed a normal cornea, anterior chamber, and lens. A completely detached retina was seen behind the lens.

In the left eye, the distance between the canthi was 28 mm; the corneal diameter was 10.5 mm. There was a searching nystagmus. The pupil reacted directly to light, and the nystagmus increased during illumination. There were doll's eye movements in all directions except abduction. Slit-lamp examination showed normal conditions.

Ophthalmoscopy revealed a myopia of 25 diopters. The disk was pale, probably slightly atrophic. The foveolar reflex was absent and the macula was

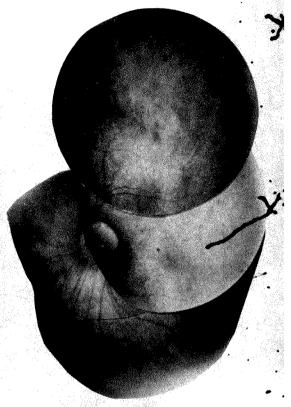


Fig. 4 (Warburg). Hydrocephaly and retinal detachment. In the larger eye there was a falciform fold and the disk was dragged vertically. Except for the 12 o'clock and 6 to 8 o'clock parts of the fundus, the retina was supplied with few vessels.

grayish brown. The rest of the fundus was light and the choroidal vessels were easily seen. The retinal vessels converged at the 6 to 8 o'clock and the 12 o'clock positions of the disk, thus leaving the larger part of the fundus devoid of vessels. In the lower nasal quadrant, the vessels followed a slightly elevated retinal fold (Fig. 4).

DISCUSSION

The patient was born at term, but was small for dates. He was given no oxygen. He had a communicative hydrocephaly, right microphthalmia with congenital retinal nonattachment, and a left abortive falciform fold. He was almost blind and severely mentally retarded.

Microphthalmia and hydrocephaly are seen in children with congenital toxoplasmosis, but this boy did not show the characteristic chorioretinal scars; nor had he any serologic evidence of toxoplasmosis.

The parents were consanguineous. In a patient with a rare malformation, this strongly suggests the presence of the homozygous state of an autosomal recessive condition.

Autosomal recessive microphthalmia in mentally retarded individuals has been discussed previously.1 It is found in patients with the Sjogren-Larsson syndrome,4 which commonly includes colebomata; in the cryptophthalmia syndrome 5,6 in which syndaetyly and urogenital malformations are also seen; in a family reported by Cross, McKusick, and Breen⁷ with cutaneous hypopigmentations and spastic palsy; and in the syndrome originally described by Saraux and associates,8 which is characterized by mental retardation, microphthalmia, congenital detachment, and osteoporosis.9-12 None of these syndromes included hydrocephaly. Our patient had some features in common with Saraux's syndrome, but he did not show the progressive nature of this disorder; nor did he have fractures.

Congenital nonattachment of the retina and congenital falciform folds are also genetic heterogeneous conditions, which can be divided into ocular and complex syndromes.² The ocular syndromes have been described both with monocular and binocular expression. In some families, the patients show either falciform folds or retinal detachment, but these two features may also occur in the same family. Both autosomal dominant^{13,14} and autosomal recessive traits have been described.^{15–19} The present patient evidently had a complex syndrome differing from the simple ocular syndromes.

Complex syndromes with congenital retinal nonattachment and congenital falciform folds have been reported in a number of autosomal recessive traits, such as the Meckel syndrome, ^{20–26} a disorder

with malformations of the central nervous system including occipital encephalocele, cleft palate, polydactyly, cysts of the liver and kidneys, genital malformations, and microphthalmia.

Furthermore, congenital nonattachment has been found in a syndrome with microcephaly and microphthalmia, 27-29 in which the affected eyes may also exhibit congenital cataract and corneal opacities. Additionally, congenital retinal detachment occurs in Saraux's syndrome. 8 Children with the Meckel syndrome usually do not survive infancy; their signs differ from those found in the patient considered here, who also showed signs different from those characterizing the microcephaly-complicated microphthalmia syndrome.

A number of complex syndromes with congenital nonattachment of the retina and X-chromosome-linked inheritance are also known, such as the Bloch-Sulzberger type of incontinentia pigmenti³⁰ and the Norrie syndrome. ^{31,32} But heredity in the present patient was not X-chromosome-linked.

Retrolental fibroplasia is the accepted diagnosis of folds or detachment of the retina in premature children treated with oxygen. Malformations with similar morphology have been reported several times in full-term infants who had not received oxygen treatment. Whether or not retrolental fibroplasia can occur without oxygen treatment is a much-debated question. Surveys of authors accepting a diagnosis of retrolental fibroplasia without prior administration of oxygen^{33,34} indicate some of their patients had symmetrical bilateral folds running from the nerve head to the temporal retina. They also had noncontributory family histories.

Servi, Giorgetti, and Cardeti³⁸ studied three sporadic cases of falciform fold and retinal detachment in premature children who had not been given oxygen. These authors accepted the suggestions of Weve¹⁷ and Mann³⁶ that such anomalies are sometimes congenital malformations and that both folds and detachments can occur in the same family. Stefani and Ehalt³⁷ analyzed 15 enucleated eyes with retinitis proliferans. In five patients, the condition was unilateral. None of the infants had been given oxygen. In their discussion of the diagnostic possibilities. these authors found that results of histologic examinations usually suggested no confident diagnosis, and that "vascular proliferation was a nonspecific secondary reaction occurring not only in retrolental fibroplasia, but also in a number of pathologic conditions wherein retinal detachment was a common feature."

In small human families, autosomal recessive disorders will often be manifested as single, or sporadic, cases. In full-term infants with retinal folds or detachments, the homozygous state of one of the previously mentioned recessive disorders should be considered. Autosomal recessive falciform folds is the diagnosis of choice in sporadic cases with symmetrical malformations, such as those reported by Brockhurst and Chishti. Sporadic occurrence of recognizable complex manifestations can also be differentiated from retrolental fibroplasia. Our patient falls into this category.

Retinal detachment in a child can be the end result of Coat's disease, but here microphthalmia and hydrocephaly are not seen; the same is true of X-chromosome-linked retinoschisis. Massive retinal fibrosis and extensive retinal hemorrhage, such as seen in the battered-baby syndrome or after other traumas, was unsupported by the general examination of our patient.

Fifteen patients with hydrocephaly, microphthalmia, retinal detachment or falciform folds have been previously described (Table).^{3,38-45} In some patients, histologic examination showed retinal dysplasia; vitreous hemorrhage was noted

in five patients, and glaucoma in two Birth weights of five of the patients were below 3,000 g; four had normal birth weight, and no information was available on the rest. No oxygen had been given, but some of the infants were suspected to have retrolental fibroplasia. The syndrome was tentatively delineated by Warburg.1,2 Svedbergh,3 in a presentation of two patients with this syndrome, proposed to call it congenital encephaloophthalmic dysplasia, the name offered. by Krause⁴¹ for a heterogenous group of disorders reported in 1946. However this designation does not take into account that many specific congenital disorders can occur simultaneously in the brain and eve, and that this particular syndrome is characterized by hydrocephaly, microphthalmia, and detachment. Svedbergh found evidence that cases of infants with hydrencephaly and proliferative retinopathy might be extreme expressions of the. same disorder. He included microcephaly and retinal detachment in this group and suggested anencephalic fetuses with retinal proliferations belong to the same group of disorders. This is doubtful.

When this syndrome was tentatively delineated in 1971, there was no evidence of whether it had a teratologic or genetic cause. This finding of the syndrome in the child of consanguineous parents indicates an autosomal recessive trait and a new nosological entity.

Evidence shows that patients with congenital hydrocephaly, microphthalmia, retinal detachment, or retinal falciform folds suffer from a rare autosomal recessive trait, not formerly delineated. Parents with a child thus affected will therefore have a 25% risk of having similarly affected children in each pregnancy.

The frequency of the syndrome is unknown, partly because patients in institutions for the mentally retarded have ophthalmic examinations only irregularly and partly because this syndrome, as well as

HMENT OR FALCIFORM FOLDS REPORTED CASES OF HYDROCEPHALUS, MICROPHITHALMIA, AND RETUNAL DETA

Authors	Case Sex No.,	Hydro- cephalus	Birth Weight (g)	Microph- thalmia	Glaucoma or buph- thalmos	Congenital Detachment	Vitreous Hemorrhage	₽НР∨*
Bernheimer ³⁶ Rochon-Duvigneau and Coutela ³⁷ Snell ³⁸	9, F. F.	+++	Fullterm	Both eyes		Both eyes Both eyes R.E. (Retinal		Both eyes L.E. Falciform
Kraiise ³⁵	6.4. P.P.C.	+++-	2,840 3,410 3,565	L.E. Both eyes R.E.	LE	dysplasia) L.E. Both eyes R.E.	Hi I	detachmen
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Reese and Straatsma*0 Laval and Chatzinoff*1 Karlsberg, Green, and Patz*6	हें ही म सन्दर्	+ +++	1,975 2,100	저 된	Neo- vascular-	Retinal dysplasia + R.E. Both eyes	Both eyes	
Svedbergh ² Present case	-, 9, F.M.M.	ተ/ሞ ተ	1,360 4,950 2,800	+ ⁰ +	ization of retina Both eyes No No	Both eyes Both eyes Right eye	No.	Falciform detachment 1. F

*PHPV, persistent hyperplastic primary vitreous. †This patient had vestigal cerebellum, cleft palate, and micrognathia.

other syndromes with falciform folds or congenital detachment, are often regarded as retrolental fibroplasia. Hereditary disorders simultating retrolental fibroplasia should be suspected in infants who have been given no oxygen, particularly in binocular and symmetrical affections. Otherwise, appropriate genetic counselling cannot be offered.

SUMMARY

A 6-year-old boy, whose parents were first cousins, had congenital retinal non-attachment in one eye and a falciform fold in the other. He had had a shunt operation for hydrocephaly. Oxygen was never administered and test results for rubella and toxoplasmosis were negative. The consanguinity in this case indicates the syndrome is an autosomal recessive trait. This and other hereditary disorders with congenital retinal nonattachment have previously been misinterpreted as retrolental fibroplasia occurring without oxygen treatment.

ACKNOWLEDGMENT

Margareta Mikkelsen, M.D., from the John F. Kennedy Institute, Glostrup, Denmark, performed the chromosome analysis.

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INFERIOR OBLIQUE MUSCLE RECESSION

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Surgical weakening of the overacting inferior oblique muscle by recession is a popular technique. Parks, 2-4 in a controlled study comparing the recession, disinsertion, and myectomy operations, found the recession operation to be the most effective and long lasting. Many techniques to localize and quantitate the recession site have been described.5-14 One of the most popular and reproducible is the Fink¹⁵ procedure, using the lateral rectus muscle as a reference point for an 8-mm recession of the inferior oblique muscle. Because of technical and exposure difficulties when using the lateral rectus muscle as the landmark in the Fink procedure, interest has developed in the use of the inferior rectus muscle instead. The present study is designed to determine the anatomy of the inferior oblique muscle in relation to the inferior rectus muscle with special reference to the surgical anatomy of the 8-mm recession site used in the Fink procedure.

MATERIAL AND METHODS

We studied 200 consecutive adult autopsy eyes, which were undeformed and without volume change. All measurements were made by using a Vernier caliper and dissecting microscope (x7). The 8-mm recession point was determined by Fink's technique. 15 The Fink localizer, an instrument consisting of two prongs, each 6 mm in length, arranged at 90 degrees to each other, was placed with one tip at the inferior border of the lateral rectus muscle insertion, following the line of inser-

tion. The tip of the second prong, extending posteriorly, automatically defined a point 8 mm from the anterior insertion of the inferior oblique muscle in the normal line of action of this muscle. We refer to this site as the "Fink point." This point was verified by measuring 8 mm from the anterior insertion of the inferior oblique muscle with the caliper. The distance of the Fink point from the lateral border of the inferior rectus muscle was then measured in two ways: first, posteriorly along the lateral border of the inferior rectus muscle and laterally (on a line perpendicular to the lateral border of the inferior rectus muscle) to the Fink point; second, laterally from the lateral insertion of the inferior rectus muscle on a line parallel to the corneoscleral limbus and posteriorly (on a line perpendicular to the corneo. scleral limbus) to the Fink point.

Additional measurements pertaining to the surgical anatomy of the inferior oblique muscle and inferior rectus muscle determined the distance between an imaginary line representing the lateral extension of the medial two thirds of the insertion of the inferior rectus muscle (the "pseudoinsertion point") and the true lateral insertion of the inferior rectus muscle.

For years, H.G. Scheie, M.D., (personal communication, March 1977) placed the anterior border of the recessed inferior oblique muscle 3 mm posteriorly along the lateral border of the inferior rectus muscle and then 2 mm laterally, but he never published the technique. In recent years, Parks^{2–3} adopted this method and described it. He considered the location an 8-mm recession. We, therefore, refer to this site as the "Scheie-Parks point." The distance of the Scheie-Parks point from the Fink point was measured.

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• tex vein in relation to the inferior oblique muscle and inferior rectus muscle was studied. We determined the distance between the anterior insertion of the inferior oblique muscle and the point at which it crossed the inferior rectus muscle. Examination for the proper location of the posterior border of the recessed inferior oblique muscle also was made. We statistically analyzed the data to determine the mean and standard deviation.

RESULTS

We determined the distance between the lateral insertion of the inferior rectus muscle and the 8-mm inferior oblique recession site (Fink point) by two methods (Figure). By method 1, the Fink point was 4.0 ± 0.7 mm posterior and 4.4 ± 0.8 mm superior to the true insertion of the lateral border of the inferior rectus muscle; it was 5.3 ± 0.7 mm posterior to the inferior rectus if the pseudoinsertion point was used. By method 2, the Fink point was 2.9 ± 0.7 mm superior (on a line parallel to the corneoscleral limbus) and 5.1 ± 0.6 mm posterior (on a line perpendicular to the corneoscleral limbus) to the true inferior rectus muscle insertion; this posterior measurement became 6.4 ± 0.6 mm if the pseudoinsertion point was used as a landmark.

The Scheie-Parks point was 2.4 mm inferior and 1.0 mm medial to the Fink point. The distance between these two points on a straight line was 3.4 ± 0.8 mm

The inferotemporal vortex vein was 11.1 ± 2.0 mm posterior to the inferior rectus muscle true insertion, and 9.9 ± 1.2 mm inferior to the posterior insertion of the inferior oblique muscle.

The distance between the insertion of the inferior oblique muscle to the point where it crosses the lateral border of the inferior rectus muscle was 13.0 ± 1.0 mm. The inferior oblique muscle insertion line was approximately parallel to the lateral border of the inferior rectus muscle.

DISCUSSION

Historically, recession of the inferior oblique muscle by using the inferior rectus muscle as a reference point is a fairly recent development. One of us (L.A.) has used this technique since about 1965, with measurements obtained at surgery similar to those obtained in this study. H.

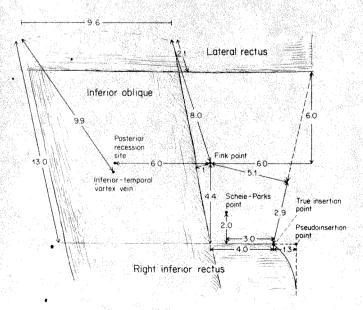


Figure (Apt and Call). Right eye, Anatomical landmarks relating recession of the inferior oblique muscle to the lateral and inferior rectus muscles. Measurements are in millimeters

G. Scheie, M.D., (personal communication, March 1977) for some 20 years, without our knowledge, had used a point 3 mm posterior and 2 mm lateral to the lateral insertion of the inferior rectus muscle to define "about a 9 - or 10-mm recession." Parks^{2,3} described and popularized Scheie's measurements and used them to obtain an 8-mm recession.

The present study defines anatomically the proper point at which the anterior edge of the inferior oblique muscle should be reinserted for an 8-mm recession, using the lateral border of the inferior rectus muscle as the reference point. We used two different methods.

In method 1 we measured posteriorly along the lateral border of the inferior rectus muscle 4.0 mm and then laterally 4.4 mm. In method 2 we measured superiorly on a line parallel to the corneoscleral limbus (also defined by a line joining the insertions of the inferior rectus muscle and lateral rectus muscle) 2.9 mm and then posteriorly 5.1 mm (on a line perpendicular to the corneoscleral limbus).

Each method has its advantages. The landmarks are better defined in method I, but the exposure is better (more anterior) with method 2.

Each technique requires careful consideration of the anatomy of the insertion of the inferior rectus muscle. Insertion is relatively straight and parallel to the corneoscleral limbus in the medial two thirds of its length. The lateral one third, however, dips posteriorly, such that the lateral border of the insertion averages 1.3 mm posterior to the medial one third (Figure). Surgical observation shows that traction by a muscle hook under the insertion of the inferior rectus muscle gives the impression that this insertion is straight, pulling the lateral one third forward into a line with the medial two thirds and defining a point we have called "pseudoinsertion" of the lateral border of the inferior rectus muscle. Measurements taken from the pseudoinsertion then must be altered by the addition of 1.3 mm to posterior dimension.

A variety of surgical procedures for weakening the inferior oblique muscle have been described, including myectomy, myotomy, disinsertion, and lecession, with variations and different techniques for each procedure!.16-19 A large prospective study of various procedures showed that recession was the most effective method to prevent return of inferior oblique muscle overaction.2,3 In that. study, the Scheie-Parks point (determined by measuring 3 mm posteriorly and 2 mm laterally from the lateral border of the inferior rectus muscle) was used to define the 8-mm recession site. Our measurements, however, show that the Scheie-. Parks point is 2.4 mm inferior and 1.0 mm medial to Fink's 8-mm recession site (3.4) mm on a straight line) (Figure). Thus, the Scheie-Parks point represents a 10.4-mm recession and a 1.0-mm anterior displacement of the inferior oblique muscle. The number of postoperative underactions (4%), described in the previously mentioned study,3 is consistent with this degree of recession. Parks recently indicated (personal communication, March 1977) he now considers his measurement. about 10 mm rather than an 8-mm recession.

Recession techniques—Ever since recession of the inferior oblique muscle was first proposed by White, 20,21 many techniques have been described. Gillies recommends measuring along the anterior border of the inferior oblique muscle during surgery, and grading the recession according to the degree of overaction. Primarily because of exposure problems, this technique can be technically difficult; additionally, rotation of the globe during surgery may alter the relationship of the inferior oblique muscle with the sclera and make measurements invalid.

Crawford⁶ determines the 8-mm recession site by reattaching the muscle just superior to the inferior temporal vortex

vein. Nutt¹¹ also uses this vein as a landmark for recession procedures. Fink²² described this vein as being 8 mm below the inferior oblique muscle insertion, but 6 mm behind the equator (1 mm anterior to the posterior border of the inferior oblique muscle). Our measurements have shown the wide variability of the location of this vein, from the insertion of the inferior oblique muscle and from the equator. Using the inferior temporal vortex vein as a reference point, then, would give posterior reinsertions and variable degrees of recession.

For a 10-mm recession of the inferior oblique muscle, Crawford⁶ recommends reinserting it at the junction where it crosses the lateral border of the inferior rectus muscle. Fink²² describes this distance as being 15 to 16 mm. Our studies show (using cord lengths) this distance averages 13 ± 1.0 mm. The discrepancy between our figures and those of Fink may represent, in part, the difference between cord and are lengths, although we were not told which method Fink used in his measurements.

Bedrossian⁵ recommends using the anterior border of the insertion of the inferior oblique muscle as a reference point, measuring I mm anteriorly for each 2 mm of inferior recession. Since the inferior oblique muscle moves posteriorly only 3.3 mm from the lateral border of the inferior rectus muscle to its insertion, this technique describes a recession plus an anterior displacement, although the described intent was to stay in the plane of the muscle action. This technique requires an adequate recognition of the anteroposterior axis of the eye, which is often difficult during surgery.

Fink's technique of using the inferior border of the lateral rectus muscle is based on extensive anatomical studies. More accurate, more reproducible, and technically simpler than those previously described, his technique has been popular for many years. However, the Fink

point used in our measurements is actually about 1 mm anterior to the normal plane of action of the inferior oblique muscle. Fink²² describes a 3.3-mm posterior movement in the 13-mm distance between the lateral border of the inferior rectus muscle and the insertion of the inferior oblique muscle, implying a 2.0+ mm anterior displacement for an 8-mm inferior oblique muscle recession. The Fink marker,22 however, allows a 3.0+ mm anterior displacement for the 8-mm recession. We based our measurements on the Fink point. The true anatomical location of the 8-mm recession site can be obtained easily by adding I mm to the posterior measurement (Figure).

The Fink recession technique presents technical difficulties. For this reason, some surgeons prefer to perform the disinsertion operation. When concomitant lateral rectus muscle surgery is not performed, it is inconvenient to expose the entire lateral rectus muscle insertion to use the Fink marker. Even when the lateral rectus muscle insertion is exposed, lining up the Fink marker and marking the 8-mm recession point is awkward, because the Fink point is difficult to approach in the lower temporal portion of the globe. Since a better exposure of the inferior oblique recession site (Fink point) is obtained with traction on the inferior rectus muscle, it is convenient and technically simpler to use this muscle as a landmark.

Anterior displacement of the insertion of the inferior oblique muscle either alone or in combination with recession, has been described as a weakening procedure. 9.23.24 Results, however, have been unpredictable.

Posterior border reinsertion—Placement of the posterior border of the inferior oblique muscle during a recession operation merits consideration since it has been given little attention in published reports. Fink¹⁵ describes reattaching the posterior border of the inferior

oblique muscle 6 mm posterior to the recessed anterior border of the inferior oblique muscle, on a line parallel to the lateral border of the inferior rectus muscle. Our measurements confirm that this recommendation is anatomically correct and surgically feasible. However, this posterior reinsertion site is often close to the intraseleral or the extraseleral, or both, extensions of the inferior temporal vortex vein; care must be taken to avoid them.

Parks,²⁻³ reinserts the posterior tip of the disinserted inferior oblique muscle 5 to 6 mm laterally and 1 mm behind the reinserted anterior tip of the inferior oblique muscle. According to our meassurments, the posterior tip of the inferior oblique muscle has been recessed 8 mm. Placement of the suture in Parks' technique for the posterior border of the disinserted inferior oblique muscle is technically simpler, but anatomically, it does not conform to the plane of muscle action; that is, the anterior tip has been recessed 10.4 mm but the posterior tip only 8 mm.

To enhance the weakening effect of the recessed inferior oblique muscle, one of us (L.A.) often bunches up the posterior border of the recessed inferior oblique muscle toward the anterior border, using one scleral suture for reattaching the muscle. This technique not only further weakens the inferior oblique muscle, but

also reduces the excyclotorsion action of the muscle by displacing the posterior • portion of the muscle anteriorly.

Graded recessions—Although a recession of 8 mm is adequate for most cases of overaction of the inferior oblique mulcle, graded recessions of a greater or lesser amount are sometimes indicated. The study by Parks, 2.3 in which the same amount of recession was done for all degrees of preoperative overaction, indicated an increased incidence of residual overaction the greater the degree of preoperative overaction, and a greater probability of postoperative underaction in eyes with a lesser preoperative overaction.

The technique described in our study. can be easily adapted to graded recessions. Our measurements indicate that the anatomical position of the inferior oblique muscle represents a 1-mm anterior displacement for each 4 mm of recession (Figure). Because the Fink point is 1 mm anterior to the true anatomical position of the inferior oblique muscle, any recession greater than 8 mm can be on a line perpendicular to the lateral border of the inferior rectus muscle, and can be easily calculated (Table). The same is true for recessions less than 8 mm, although to be anatomically accurate, measurements should be 1 mm posterior to the Fink point (Table).

TABLE
RECOMMENDED MEASUREMENTS FOR GRADED RECESSIONS OF THE INFERIOR OBLIQUE MUSCLE

Desired A	mount of Recession mm	(From la	ed Posteriorly teral border of insertion) (mm)	(Perr	asured Superiorly pendicular to lateral rder of IR*) (mm)
	6 7 8		5.0 5.0 4.0		6.4 5.4 4.4
	10 11 12		4.0 4.0 4.0		2.4 1.4 0.4

^{*}IR indicates inferior rectus muscle.

SUMMARY

Surgical recession of the inferior oblique muscle is simpler to perform if the inferior rectus muscle, rather than either the lateral rectus muscle or the inferior oblique muscle insertion, is used as a landmark. We measured 200 consecutive autopsy eyes to determine the distance from the commonly used 8-mm recession site determined by the Fink · technique to the lateral border of the inferior rectus muscle insertion. It was easier to reach this point by measuring 4.0 mm posterior and 4.4 mm superior to the lateral insertion of the inferior rectus muscle, or 2.9 mm superior (on a line parallel to the corneoscleral limbus) and 5.1 mm posterior (on the line perpendicular to the corneoscleral limbus) to the lateral insertion of the inferior rectus muscle.

.We made anatomical studies to grade the amount of inferior oblique muscle recession and to evaluate the proper placement of the posterior border of the recessed inferior oblique muscle.

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CONJUNCTIVAL RESECTION TREATMENT AND ULTRASTRUCTURAL HISTOPATHOLOGY OF SUPERIOR LIMBIC KERATOCONJUNCTIVITIS

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Theodore¹ first described superior limbic keratoconjunctivitis in 1963. It is characterized by inflammation of the superior tarsal and bulbar conjunctiva and edema of the corneoscleral limbal conjunctiva; corneal filaments are frequently present. Fine punctate fluorescein and rose bengal staining of the superior cornea are commonly found. This condition is usually bilateral, occurs most frequently in females, and has been reported in patients from ages 4 to 81 years.2 Superior limbic keratoconjunctivitis characteristically follows a chronic course with remissions and exacerbations. The prognosis most often is excellent in that eventual resolution occurs without visual impairment.

Laboratory studies in the form of bacterial, viral, or fungal cultures do not help determine diagnosis, cause, or treatment.² The diagnosis can often be aided by finding keratinized epithelial cells with degenerated nuclei and hyalinized cytoplasm on smears from scrapings of the involved bulbar conjunctiva.³

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An association with thyroid disease has. been reported. Tenzel4 was the first to draw attention to the presence of elevated protein-bound iodine in patients with superior limbic keratoconjunctivitis. Cher⁵ reported that five of his ten patients were either thyrotoxic or had undergone treatment for hyperthyroidism at the time they had superior limbic keratoconjunctivitis. Similarly, Wright⁶ noted an incidence of 26% of present or past thyroid disease in his patients with superior limbic keratoconjunctivitis. Fells⁷ stated that 40% of his patients with dysthyroid exophthalmos had superior limbic keratoconjunctivitis. Theodore⁸ reported the presence of hypothyroidism in some patients with superior limbic keratoconjunctivitis.

The clinical benefits of 0.5% silver nitrate application to the palpebral conjunctiva of the upper and lower eyelids of superior limbic keratoconjunctivitis patients have been advanced by Theodore.9 Other authors, 5.6 not finding silver nitrate therapy effective, have suggested mechanical removal of the surface cells, topical use of N-acetylcysteine, 1% adrenalin, and soft contact lenses. The successful results of these various forms of therapy, however, have rarely been dramatic or long lasting. Tenzel10 has reported that recessing the superior limbic conjunctiva resulted in the elimination of symptoms. He also found that resection of the conjunctiva with suturing of the free edges to the episclera resulted in long-lasting improvement of symptoms (R. R. Tenzel, M.D., personal communication, 1976).

The current study was undertaken to investigate the effect of palpebral or bulbar conjunctival resection on the signs and symptoms of superior limbic keratoconjunctivitis and to document, for the first time known to us, the essential ultrastructural histopathologic features demonstrated in ocular tissues affected by this disorder. Since a viral cause is often postulated in diseases of unknown origin, we conducted a specific search for viral particles in the excised tissue.

CASE REPORTS

Case 1—A 56-year-old white woman was examined because of intermittent, disabling, bilateral ocular irritation of ten years' duration. Results of

thyroid T₄ and T₄ tests were normal.

• Examination revealed a corrected visual acuity of 6/6 (20/20) in each eye. Results of the ocular examination were unremarkable except for the superior limbic area of both eyes. Mild injection of the conjunctival vessels was present bilaterally. The superior limbic conjunctiva appeared congested, edematous, and thickened. Moderate punctate staining with rose bengal was demonstrated over the superior conjunctiva and superior corneal epithe—
Tium. The tarsal conjunctiva showed fine bilateral papillary inflammatory changes. Results of Schirmer tests were normal.

Over the ensuing five months, trials of artificial tears, topical corticosteroid therapy, soft contact lenses, and 0.5% silver nitrate applications to the superior tarsal conjunctiva were entirely ineffective.

An excision of bulbar conjunctiva of the right eye was performed with immediate relief of symptoms. Postoperative recovery was uneventful. Three weeks after conjunctival resection, examination revealed a quiet, asymptomatic right eye with a smooth limbal conjunctiva, without rose bengal staining. The untreated symptomatic left eye exhibited impressive rose bengal superior limbic conjunctival staining, ·with congested conjunctival tissue exhibiting a typical heaped-up, corrugated appearance adjacent to the corneoscleral limbus. One month later an excision of bulbar conjunctiva of the left eye was performed. Follow-up examinations six and five months after the respective resections showed continued improvement. The only complaint was occasional irritation and the presence of excessive mucus. Ocular examination revealed normal eyes, except for a slight papillary tarsal conjunctival response. The superior corneoscleral limbus was smooth and it did not stain with rose bengal.

Case 2—A 56-year-old white woman was referred with a history of bilateral superior limbic keratoconjunctivitis of three years' duration. T₃ and T₄ studies were normal. She had been unsuccessfully treated with topical corticosteroids, decongestants, antibiot-

ics, artificial tears, and lubricant ointments. Examination demonstrated all the clinical features of superior limbic keratoconjunctivitis, including bilateral corneal filaments. Schirmer test results were normal.

A soft bandage contact lens was applied to the right eye with subsequent relief of symptoms and elimination of filaments. The untreated left eye remained objectively, as well as subjectively, unchanged.

The soft lens was removed from the right eye and a similar lens was applied to the left one. The right eye became symptomatic and corneal filaments developed. The superior limbic conjunctiva was again thickened and congested, and rose bengal staining was present. The left eye, under the soft contact lens, was symptomatically improved but objectively

unchanged.

We resected the bulbar conjunctiva of the right side. The soft contact lens was removed from the left eve. After resection, the patient experienced relief of symptoms in the right eye, but the left eye remained irritated and unchanged for six weeks after removal of the contact lens. A resection of the superior tarsal conjunctiva of the left eye was performed. Postoperatively no rose bengal staining was present in either eye. The tarsal conjunctival papillary reaction was still present in the right eye, but the superior tarsal conjunctival tissue of the left eye appeared normal. Seven months after resection of the conjunctiva, the right eve was asymptomatic with a smooth superior corneoscleral limbus and no evidence of fluorescein or rose bengal staining. The left eye (tarsal conjunctival resection) was symptomatic and filaments had recurred.

Case 3—This 58-year-old woman complained of a slowly progressive, painless decrease in vision in the right eye, accompanied by ocular irritation in both eyes. Ophthalmoscopic examination revealed a corrected visual acuity of R.E.: 6/12 (20/40), and L.E.: 6/120 (20/400). She had a 10-diopter myopia and a large astigmatic error of 15 diopters in the left eye. Ocular examination revealed mild bilateral superior conjunctival features of superior limbic keratoconjunctivitis. Elevated T3 and T4 values were noted which required subsequent hospitalization and treatment with radioactive iodine. For the next five months, the patient received artificial tears for her complaints of ocular irritation; her clinical appearance remained unchanged. A resection of the superior limbic conjunctiva was performed with immediate relief of symptoms. Six months after resection she has remained asymptomatic although slight superior conjunctival injection has remained.

Case 4—A 40-year-old woman was referred with a 3½-year history of recurrent ocular irritation, with filaments and corneal ulcers of the right eye. She had been treated with a variety of drops and ointments including silver nitrate. Thyroid function studies

were reported to be normal.

Corrected visual acuity was R.E.: 6/9 (20/30), L.E.: 6/6 (20/20). Mild limbal conjunctival thickening and injection of the superior corneoscleral limbus was noted. Superficial punctate staining of the

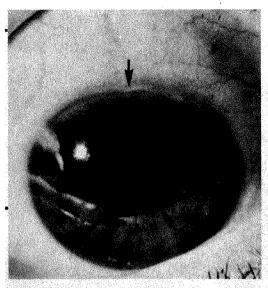


Fig. 1 (Donshik and associates). Case 4. Clinical photograph showing limbal epithelial thickening (arrow) and injection.

cornea was present, but filaments were absent (Fig. 1). Schirmer test results were normal. The left eye was unremarkable. Treatment consisting of artificial tears every two hours gave no improvement. The superior limbal conjunctiva was then resected and the patient experienced immediate relief. After resection the corneoscleral limbus was smooth and flat. The patient has remained totally asymptomatic for ten months.

METHODS

The bulbar conjunctiva (from one eye in three patients and from both eyes in a fourth patient) was surgically resected under local anesthesia. A block of conjunctiva extending from the 10:30 to the 1:30 position at the corneoscleral limbus and posteriorly for 3 to 4 mm was routinely removed. Erythromycin ointment was instilled and a patch was applied for 48 hours.

In one patient approximately 2.5 mm of the upper tarsal conjunctiva of one eye was resected. Erythromycin ointment was instilled and a patch was worn for 24 hours. No surgical complications were encountered, and healing was prompt and complete. We performed light and transmission electron microscopy on the five samples of superior bulbar conjunctiva and on the one sample of superior tarsal conjunctiva. Controls consisted of similar tissue from patients who underwent cataract surgery.

On removal, tissue for electron migroscopy was immediately placed in buffered glutaraldehyde (4% in 0.067M sodium cacodylate at pH 7.35) at room temperature. Tissue was dissected into small pieces and processed in one of the following ways:

(1) Normal osmium—Tissue was fixed for one hour in 2% osmium tetroxide in 0.067M sodium cacodylate, dehydrated in acetone, and then embedded in Araldite. Uranyl acetate (0.5% in 75% acetone) block stain was used and sections were stained with lead citrate¹¹ and uranyl acetate (1% aqueous).

(2) Osmium potassium ferrocyanide—Fixation was accomplished with 2% osmium tetroxide and 1.5% potassium ferrocyanide in 0.067M sodium cacodylate for one hour. This is a slight modification of the osmium potassium ferrocyanide method of Dvorak and co-workers. Tissue was then dehydrated in acetone and embedded in Araldite. Sections were stained with lead citrate only. Sections were cut on an LKB Ultratome III, and examined on an electron microscope.

Sections 1µ thick were stained with Richardson's stain. Paraffin sections were also prepared and routinely stained with hematoxylin and eosin for light microscopy.

RESULTS

Light microscopy—Limbal conjunctiva of control cases demonstrated no signs of keratinization, no intracellular swelling, and no signs of inflammation. Limbal conjunctiva that was removed from all superior limbic keratoconjunctivitis patients demonstrated various degrees of keratinization with areas of acanthosis.

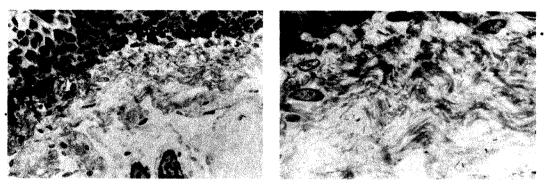


Fig. 2 (Donshik and associates). Case 2. Light micrograph of epithelium and stroma (left) and stroma (right) of the lower corneoscleral limbal conjunctiva showing blood vessels packed with erythrocytes, dilated lymphatic vessels, and stromal tissue that is relatively devoid of inflammatory cells (Richardson's stain, left, ×385; right, ×960).

Some epithelial cells appeared swollen and in various stages of degeneration. In particular, the nuclei of some cells appeared empty. The stroma showed signs of edema, with grossly dilated lymphatic

vessels. Inflammatory cells-neutrophils, lymphocytes, and plasma cells-were rare and could not be distinguished from cells of normal controls (Fig. 2). Tarsal conjunctiva from one patient revealed an

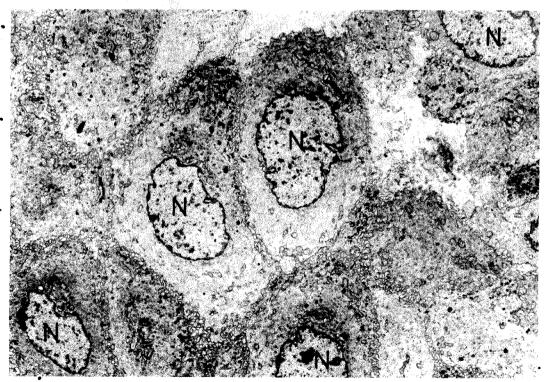


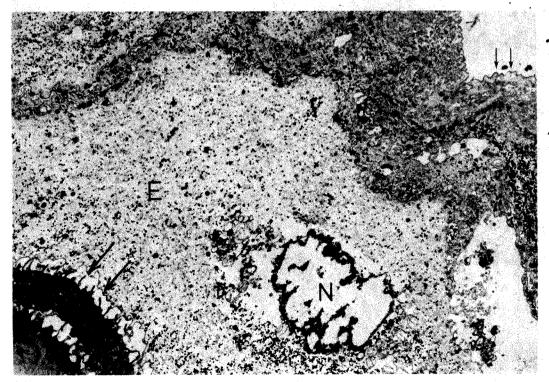
Fig. 3 (Donshik and associates). Superficial epithelial cells with large nuclei (N) from the superior corneoscleral limbal region of a normal eye. Tissue was removed during cataract surgery (osmium potassium ferrocyanide, ×3,900).

essentially normal epithelium, except for some intercellular edema, with no evidence of keratinization. The goblet cell population appeared similar to control tissue. The underlying stromal tissue showed few inflammatory cells. There were some areas of cellular debris.

Transmission electron microscopy—Superior limbal conjunctiva of control patients revealed normal epithelium with small intercellular spaces and no evidence of glycogen accumulation in the cells (Fig. 3). The most prominent feature of the superficial perilimbal bulbar conjunctiva taken from superior limbic keratoconjunctivitis patients was the consistent finding of large, pale, swollen cells in which the cytoplasmic organelles were scarce. In some cells the nuclei were large and empty, although the nuclear mem-

brane was often intact; this appeared to be a form of balloon degeneration of the nuclei. In other cells the nuclear envelope was broken and nuclear material was released into the cytoplasm. Intercellular spaces were larger than in the normal tissue and there was some breakdown of desmosomes (Fig. 4). Other nuclei also showed changes in the distribution of darkly staining nuclear chromatin, which was aggregated in the center of the nucleus, instead of around the nuclear membrane or evenly distributed as in normal cells. The nuclear envelope was also more tortuous (Fig. 5).

Another prominent feature was the presence of extensive intracellular accumulations of glycogen granules (Figs. 4 and 5), which appear black (electrondense) when the osmium potassium ferro-



* Fig. 4 (Donshik and associates). Case 4. Section of superior limbal conjunctiva. There is a large, pale, swollen cell (E) surrounded by more darkly staining epithelial cells. The nucleus (N) is empty and undergoing balloon degenerative changes. Areas of thickened cell membrane (small arrows) and breakdown of desmosomes (large arrows) are shown. The small black dots represent glycogen granules which are dense in the cells at the lower left of the micrograph (osmium potassium ferrocyanide, ×6,750).

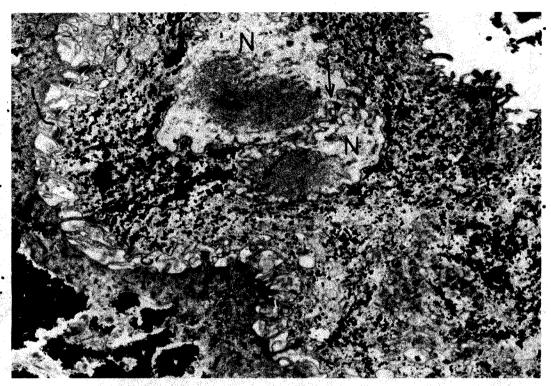


Fig. 5 (Donshik and associates). Superior limbal epithelial tissue from patient 3. The two sections of the nucleus (N) contain an unusual distribution of darkly staining chromatin. The nuclear envelope is abnormally tortuous (arrows). Black dots and areas represent abnormally accumulated glycogen (osmium potassium ferrocyanide, ×14,300).

cyanide method of fixation is used. Although superficial conjunctival epithelial cells contain some glycogen, 14 it is minimally present. Thoft and Friend 15 found only 8.8 μ M glucose/g of dry weight in rabbit conjunctiva compared to 237.1 μ M/g of dry weight in rabbit corneal tissue. We were unable to demonstrate glycogen granules in the control specimens, yet the intracellular glycogen granules were clearly visible in all the specimens from patients with superior limbic keratoconjunctivitis.

There was extensive evidence of keratinization of the epithelium (Fig. 6). This was in the form of increased numbers and clumping of microfilaments, the presence of secondary lysosomes, thickening of the epithelial cell membrane, and the presence of keratohyalin granules in the cyto-

plasm (Fig. 7). We are preparing a more complete account of the ultrastructural changes associated with accumulation of keratohyalin granules.

In patients who had been treated with silver nitrate, silver granules were deposited in the intercellular spaces. Although viral particles were diligently searched for, none could be found. Marked inflammatory cells were not present in either the epithelium or stroma of the resected conjunctiva.

Ultrastructural examination of tarsal conjunctiva taken from one patient with superior limbic keratoconjunctivitis confirmed the light microscopic findings of a relatively normal epithelium, with some edema and an occasional inflammatory cell. Glycogen granules were rare and there was no aggregation of granules. In

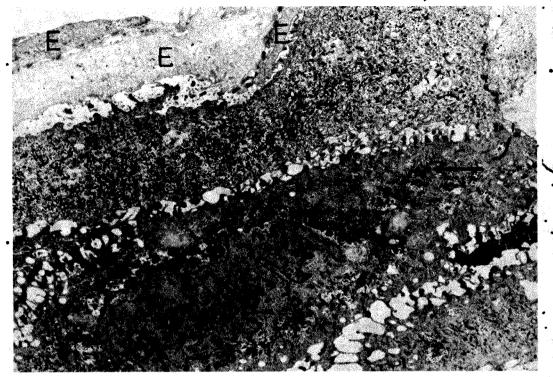


Fig. 6 (Donshik and associates). Case 4. Superior limbal conjunctiva (C) showing abnormalities of the superficial epithelial cells. The three cells (E) show loss of cell components and are becoming keratinized. The intercellular spaces are wide and the deeper cells show an increase in the number of cytoplasmic filaments. A small keratohyalin granule (arrow) is present (normal osmium, $\times 6,000$).

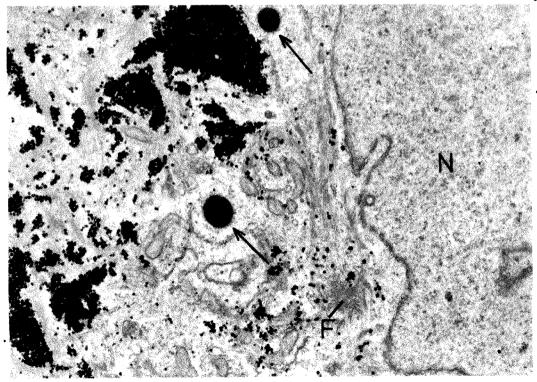


Fig. 7 (Donshik and associates). Case 4. Part of a superior limbal epithelial cell. There are two keratohyalin granules (arrows) surrounded by what appear to be ribosomes. Microfilaments are numerous (F) and glycogen (black) is present Nucleus (N) (osmium potassium ferrocyanide, $\times 18,800$).

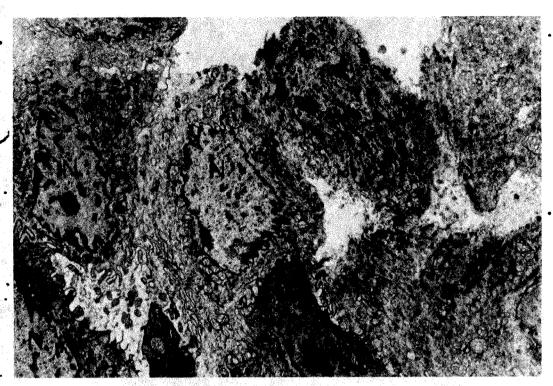


Fig. 8 (Donshik and asociates). Case 4. Superficial epithelial cells from the upper tarsal conjunctiva. The majority of cells have essentially normal nuclei (N). There are no signs of keratinization and no accumulations of glycogen granules (osmium potassium ferrocyanide, ×6,750).

the stroma there were a few disintegrating cells and some areas of cell debris, but no evidence of an inflammatory response (Fig. 8).

DISCUSSION

Previous histologic descriptions of the superior'limbic conjunctival tissue of patients with superior limbic keratoconjunctivitis have noted keratinization of the epithelium with acanthosis and dyskeratosis, as well as a moderate infiltration of inflammatory cells, chiefly neutrophils, lymphocytes, and plasma cells.3,5,6 Light microscopic examination of the tarsal conjunctiva has shown an increase of mucoid material on its surface together with a slight inflammatory cell infiltration.6 Theodore and Ferry3 reported a significant infiltration with neutrophils, lymphocytes, and plasma cells with normal epithelium.

Our studies by both light and transmission electron microscopy confirm the presence of abnormal superior limbic epithelium consisting of keratinized epithelial cells with swollen and degenerated nuclei. The stroma, although edematous, demonstrated no evidence of significant inflammatory cellular infiltrate. An inflammatory cell response in the patients studied by Theodore and Ferry may well have been secondary to prior silver nitrate treatment.

The symptoms in superior limbic keratoconjunctivitis may stem from the interaction of three abnormal elements: the elevated bulbar conjunctiva, the superior corneal epithelium, and the papillary reaction of the superior tarsal conjunctiva. The elevated bulbar conjunctiva in apposition to the tarsal conjunctiva could produce a foreign-body sensation. Furthermore, the raised bulbar conjunctiva can

prevent the tear film from adequately wetting the superior cornea, causing superior punctate desiccation and filament formation. Although the cause of superior limbic keratoconjunctivitis is unknown, therapy should be directed toward decreasing the limbal engorgement and adequately moistening the conjunctiva and cornea. This can be accomplished by mechanical debridement (scraping), chemical debridement (silver nitrate therapy), or soft contact lenses.

Wright postulated that the initial pathologic process may reside in the upper tarsal conjunctiva. He argued that the presence of a chronically inflamed upper eyelid may be responsible for a disturbance in the normal maturation of the bulbar conjunctival cells. He believed the normal upper evelid, in the presence of mucus of normal composition and viscosity, to be responsible for removing the superficial cells of the conjunctiva. If this process is impaired by an abnormality of either the evelid or mucus, then the bulbar conjunctival cells persist in situ and progress toward keratinization. Any conjunctival irritation can cause an increased goblet cell activity with increased mucus production and an alteration of the mucus-tear relationship.16

Although this is a possible explanation, we were unable to identify definitively either the superior limbal conjunctiva or the palpebral conjunctiva as the primarily involved tissue. Several observations indicate that the abnormality resides in the bulbar conjunctiva. In Case 4 the presence of papillary tarsal reaction in the right eye did not result in the reappearance of abnormal keratinization of the superior limbic conjunctiva after resection. In Case 2 removal of either palpebral or bulbar conjunctiva was enough to break the abnormal cycle and produce immediate relief. However, symptoms did return in the eye that underwent tarsal resection. Furthermore, we were unable to demonstrate any structural abnormalities or increased goblet cells in the tarsal conjunctiva.

In our cases, by removing the abnormal tissue, a normal relationship between the involved structures was re-established. Thus conjunctival resection affords long-lasting remissions of symptoms and should be considered as a therapeutic modality in patients with superior limbic keratoconjunctivitis who have either not responded to a trial of medical management or who have experienced frequent symptomatic recurrences on medical therapy.

Possible causes include dry eye, endocrine imbalance, viral involvement, and immunologic abnormalities. Our patients had normal Schirmer test results, but one was thyrotoxic. There was no histologic evidence of viral involvement. While autoimmunity is an attractive possibility, one would then expect corticosteroid therapy to have a beneficial effect, but it does not.

The possibility that the inciting agent is an abnormal quality or quantity of mucus should be considered and warrants further investigation. Two of our patients (Cases 1 and 2) had recurrence of mild symptoms in the presence of moderate discharge.

SUMMARY

Four patients with symptomatic superior limbic keratoconjunctivitis underwent resection of the superior bulbar conjunctiva. One of these patients also underwent a tarsal conjunctival resection in the other eye. Three of the patients had previously been treated by various regimens without resolution; the fourth had had no prior treatment. All four patients had immediate and continued relief of the ocular symptoms after the superior bulbar conjunctiva was excised. The patient who underwent tarsal conjunctival resection experienced only short-term relief.

We studied the conjunctival tissue by light and transmission electron microsco-

py. Both techniques revealed abnormalities related to the bulbar conjunctival surface with keratinization of the epithelium, acanthosis, degeneration of the nuclei, and intracellular accumulation of glycogen. Inflammatory cells were minimally present. The tarsal conjunctiva appeared essentially normal.

ACKNOWLEDGMENT

Deborah Pavan-Langston, M.D., and Claes Dohlman, M.D., allowed us to study their respective patients.

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NEUROPARALYTIC KERATITIS IN GOLDENHAR-GORLIN SYNDROME

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AND

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The malformations comprising the oculoauriculovertebral dysplasia complex were first described in 1845 by von Arlt, but were not recognized as a syndrome until 1952 when Goldenhar described the classical triad of epibulbar dermoids, preauricular tags and blind fistulas, and vertebral anomalies.² Associated ocular defects, facial anomalies, and skeletal malformations have also been reported.³⁻⁶

Most recently, Gorlin, Cervenka, and Pruzansky⁷ demonstrated that Goldenhar's syndrome is only a mild form or variant of hemifacial microsomia, and this contribution has been recognized⁸ by eponymic redescription of this complex as the Goldenhar-Gorlin syndrome or oculoauriculovertebral dysplasia-hemi-facial microsomia.

Unilateral neuroparalytic keratitis in this syndrome has been reported twice to date. 4.9 We report a third such case, which was bilateral, to alert clinicians to this potentially blinding problem and its cause.

CASE REPORT

A 2-year-old white boy, the product of a full-term uncomplicated pregnancy and delivery, had a birth weight of 3.5 kg and was known to have Goldenhar's syndrome and congenital esotropia. He had had red eyes for two months. The referring ophthalmologist, before therapy, took a conjunctival culture which had grown only *Staphylococcus epidermidis*. The conjunctivitis was then treated with several topical antibacterial agents without resolution. Four days before referral, corneal opacities were noted and the

patient received idoxyuridine drops for presumptive herpetic keratitis. Because of progressive ulceration in the next 48 hours, he was referred here (Fig. 1).

On admission, each eye fixed centrally. Pupillary reactions were normal. There was a moderate esotropia. The bulbar conjunctiva was markedly injected and ciliary flush was present in both eyes. An epibulbar dermoid was present at the 9 o'clock aspect of the corneoscleral limbus of the right cornea. Slit-lamp examination revealed a 4-mm central ulcer in the right cornea and a 2-mm uncer in the left cornea. There were 3+ flare and 2+ cells in both anterior chambers. The media and fundi were otherwise normal. General physical examination was within normal limits except for bilateral preauricular skin tags (Figs. 2 and 3).

On admission the topical antibacterials were discontinued and treatment was limited to topical atropine drops. The following day appropriate aerobic and anaerobic bacterial, viral, and fungal cultures were taken, as well as material for conjunctival and corneal smears. Although smears of conjunctival scrapings showd an assortment of bacteria in small numbers, smears of scrapings from the margins of the corneal ulcers showd only a few polymorphonuclear leucocytes with no inclusion bodies and no fungi

We were amazed at the ease with which we obtained these cultures and scrapings with minimal topical anesthesia. We subsequently tested the corneas on several occasions and found them to be virtually anesthetic. A Schirmer I tear test showed deficient tear production (4- and 5-mm wetting in the right and left eye, respectively).

Until all cultures were reported negative at 48 hours, the patient received subconjunctival and topical antibiotics with minimal effect. Subsequent



Fig. 1 (Mohandessan and Romano). The patient on admission, with bilateral sterile corneal ulcers that are worse on the patient's right.

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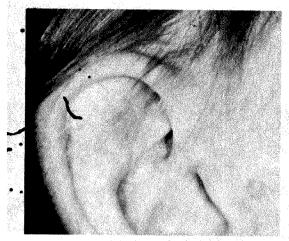


Fig. 2 (Mohandessan and Romano). The patient's right ear shows a preauricular skin tag.

treatment was limited to atropine, sterile ophthalmic petrolatum ointment, and humidification of the patient's crib. The ulcers in both eyes healed in two weeks without further treatment or problems.

. Neurologic consultation confirmed our findings of hypoesthesia limited to the ophthalmic division of the fifth cranial nerve bilaterally, without evidence of other neurologic deficit.

DISCUSSION

In addition to the two similar reported cases cited,^{4,9} one case of unilateral corneal anesthesia in Goldenhar's syndrome,



Fig. 3 (Mohandessan and Romano). The patient's left ear show multiple preauricular skin tags.

without corneal ulceration, was reported by von Bijsterveld.¹⁰

In our case and in two of the three reported cases, tear production was deficient. This is regularly seen in corneal denervation of varying causes and the resultant drying is, in turn, the cause of corneal breakdown and ulceration.¹¹

The corneal anesthesia is caused by a defect of the ophthalmic division of the trigeminal nerve. The level at which this occurs is probably nuclear and the result of a nuclear aplasia. This hypothesis is based on autopsy findings in a severe case of unilateral Goldenhar-Gorlin syndrome. The child was anophthalmic and therefore did not have a neuroparalytic keratitis. Postmortem examination of serial sections of the brain stem revealed absence of the ipsilateral sensory and motor nuclei of the trigeminal nerve.

SUMMARY

A 2-year-old boy had bilateral corneal ulceration with the Goldenhar-Gorlin syndrome. Initially, the patient received subconjunctival and topical antibiotics with minimal effect. With subsequent treatment of atropine, sterile ophthalmic petrolatum ointment, and humidification of the patient's crib, ulcers in both eyes healed in two weeks without further problems. He had a neuroparalytic keratitis with corneal anesthesia and decreased tear production bilaterally. Our evidence suggested that aplasia or hypoplasia of the trigeminal nuclei was the probable cause.

ACKNOWLEDGMENT

Nelson Gurney, M.D., referred this patient to us.

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OPHTHALMIC MINIATURE

Celsus mentions a probe which-strangely enough-was soluble: a medicated stick, called collyrium. Collyria were made up of a glutinous paste rolled into long thin cones. ...The little stick could be used to explore wounds, or they could be broken up and dissolved to make up a medicated solution. Eventually, the second use prevailed, and the resulting solutions were used mainly for the eyes.

Guido Majno, *The Healing Hand* Harvard University Press, Cambridge, Mass., 1975, p. 359

AEROMONAS HYDROPHILA CORNEAL ULCER

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Aeramonas hydrophila, a gram-negative rod, is commonly isolated from water, soil, or foods, but rarely from humans. · Recently, it has been increasingly implicated as a human pathogen, particularly in patients with an altered defense system or a traumatic wound, especially when the wound is contaminated with water or material from an aquatic environment. One case of endophthalmitis has been reported with A. hydrophila in a mixed infection. This is a report of two cases of corneal ulcer caused solely by A. hydrophila.

CASE REPORTS

Case 1—A 27-year-old healthy man had pain. · photophobia, and purulent discharge for one day after being struck in the right eye with a piece of seashell while moving the lawn. His ophthalmologist diagnosed a corneal ulcer eight hours after the initial trauma and referred him for treatment. Examination showed visual acuity of R.E.: 6/21 (20/70), and L.E.: 6/7.5 + 2 (20/25 + 2). The left eye was entirely normal. The right eyelids were covered with dry, purulent exudate. The conjunctiva was diffusely injected. A 2-mm corneal ulcer with discrete margins was in the superior cornea 3 mm from the corneoscleral limbus; the ulcer involved two thirds of the corneal stromal depth. A white infiltrate was present on the edge of the lesion and an area of marked edema surrounded the ulcer. The cornea showed diffuse epithelial edema and an hypopyon was present in the anterior chamber (Fig 1). The pupil was dilated and nonreactive from prior mydriatics. The lens and posterior eye were normal. Gram stain of the corneal ulcer scrapings contained multiple gram-negative rods. The predominant inflammatory cell type was polymorphonuclear leukocyte; some contained phagocytized bacteria (Fig 2). A diagnosis of gram-negative, bacterial corneal ulcer was made and the corneal ulcer and conjunctiva were cultured on blood agar, chocolate agar, and thioglycolate meat broth. Initial antibiotic therapy

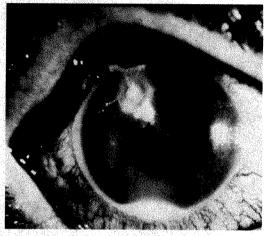


Fig. 1 (Feaster, Nisbet, and Barber). Case 1. The right eye is diffusely injected. A discrete ulcer is seen in the upper cornea. A hypopyon is present.

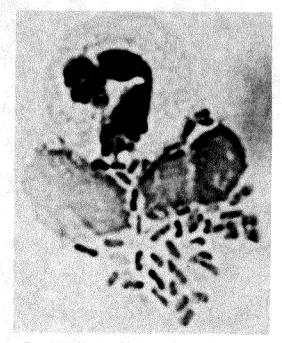


Fig. 2 (Feaster, Nisbet, and Barber). Photomicrograph of the corneal scrapings showing multiple gram-negative rods surrounding two corneal epithelial cells and a polymorphonuclear leukocyte (gram stain, \times 630).

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TABLE
Antibiotic sensitivities of Aeromonas
hydrophila cultures

	Case 1	Case 2
Ampicillin	R	R
Carbenicillin	S	R
Cephalosporin grp.	S	R
Chloramphenicol	S	S
Gentamycin	S	S 1
Kanamycin	S	S
Neomycin	\mathbf{S}	S
Nitrofurantoin	S	
Polymyxin grp.	S	S
Streptomycin grp.	S	
Sulfonamide grp.	S	
Tetracycline grp.	S	S
Tobramyein grp.	S	S

S denotes susceptible; R, resistant.

consisted of 20 mg of subconjunctival gentamycin and 80 mg of intramuscular gentamicin every eight hours. One gram of methicillin was administered intravenously every six hours. Gentamycin ophthalmic ointment was applied during waking hours and every three hours at night. Topical scopolamin 0.25% was given three times daily. The purulent discharge, corneal edema, and hypopyon decreased within 24 hours. Thirty-six hours after admission the infecting organism was identified as A. hydrophila cultured from both corneal and conjunctival specimens. Antibiotic sensitivities were tested (Table). Methicillin was discontinued and topical and systemic gentamycin continued. The ulcer rapidly improved with complete healing of the epithelium and resolution of the hypopyon over the next several days. The patient was discharged and we prescribed gentamicin ointment four times daily; he has continued to do well. Final visual acuity in the involved eye was 6/7.5 +2 (20/25 +2).

Case 2-A 30-year-old healthy man was admitted with a one-day history of left eye pain after a foreign-body injury to his left eye while he was working on an offshore oil rig. His ophthalmologist had diagnosed a corneal ulcer and referred the patient for treatment. Initial examination showed visual acuity of R.E.: 6/6 (20/20), and L.E.: finger counting at 3 feet. The right eye was entirely normal. The left conjunctiva had a diffuse, severe injection with ciliary flush. The left cornea had a 3-mm central corneal ulcer, one-half the stromal depth, with a surrounding ring of white infiltrate at the periphery of the ulcer. The epithelial defect extended beyond the ulcer margin and an hypopyon was . present (Fig 3). The lens and posterior eye were normal. Gram stain of the ulcer scrapings contained many gram-negative rods. A diagnosis of gramnegative bacterial corneal ulcer was made and the ulcer was cultured on blood agar, chocolate agar,

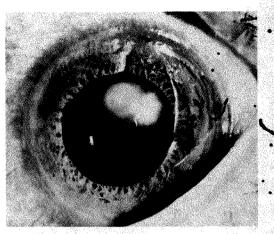


Fig. 3 (Feaster, Nisbet, and Barber). Case 2. The eye is diffusely injected. A discrete corneal ulcer is seen near the center of the cornea.

and thioglycolate meat broth. The patient was given antibiotic therapy of 20 mg of subconjunctival gentamycin, 100 mg of subconjunctival methicillin, 80 mg of gentamycin intravenously every eight hours and 2 million units of penicillin intravenously every four hours. Topical gentamycin and polymyxin B sulfate neomycin sulfate and gramicidin (Neosporin) drops were alternated hourly. Topical scopolamine 0.25% was given three times daily. The intensity of the infiltration and the hypopyon decreased within 24 hours. The infecting organism was identified as A. hydrophila 24 hours after admission. Antibiotic sensitivities were tested (Table). The penicillin was discontinued while systemic and topical gentamycin and Neosporin were maintained. The ulcer healed by the sixth hospital day with a small corneal opacity remaining. The patient was discharged and prescribed topical gentamycin, chloramphenical, and scopolamine. The patient is currently doing well with a visual acuity of 6/7.5 (20/25).

DISCUSSION

Aeromonas hydrophila is a gramnegative bacterium of the family Pseudomonaceae. It is motile, polarly flagellated and oxidase-positive. It grows on routine aerobic bacterial media and usually gives a strong beta-hemolytic reaction. It ferments carbohydrates with the production of gas or acid or both. The details of the biochemical characteristics have been outlined elsewhere.²⁻⁴ The morphologic characteristics of the bacterial colonies are not specific enough to allow visual identification and they may easily be mistaken for members of the family Enterobacteriaceae. Many sources have recommended the routine performance of the oxidase test for the screening of gramnegative cultures to prevent this confusion. 5-7

The primary habitat of A. hydrophila is stagnant water, water wells, and nonfecal sewage with a high content of organic substances which, in turn, contaminate water sources. It has been cultured from hospital water supplies and is a potential source of nosocomial infection. Slotnick observed that the survival of A. hydrophila depends on the presence of moisture and organic matrix on the surface of the material upon which it grows.

Aeromonas hydrophila has long been known to produce "black-rot" in hen eggs.9,10 It also causes diseases in marine and fresh water animals such as fish and amphibians. It can produce experimental disease in mice, guinea pigs, and rabbits.11 Although it has occasionally been cultured from the intestinal tract of asymptomatic humans, it is probably a transient inhabitant.2,4,12 It has been increasingly implicated as a human pathogen, especially in patients with compromised defense systems or in wound infections contaminated by water or soil. It is probably an opportunistic pathogen in the human. Hibbs, Merker, and Kruckenberg11 found that experimentally induced infection in rabbits was more easily induced in rabbits that were stressed by starvation. The organism has been identified in many diseases in pure culture such as intestinal distrubances, 13 peritonitis, 14 bacteremia and sepsis, 1.5.8,14-18 osteomyelitis17 and wound infections.1.18 Shulkin19summarized the basic mechanisms of infection caused by A. hydrophila as being enteric, bacteremic, or direct contamination.

There are no reports of A. hydrophila cultured from normal healthy human eve-

lids or conjunctiva. Washington reported A. hydrophila endophthalmitis in an eye which was also infected with Clostridium perfringens, Bacillus species, and diphtheroids after perforation of the eye in a dynamite explosion. No case of corneal ulcer caused solely by A. hydrophila has been reported previously. It is significant that both cases in this report had contamination of the traumatic corneal wound with material from an aquatic environment.

Antibiotic sensitivities in the cases presented paralleled those of previous reports. 1.5-7.20.21 A. hydrophila has been consistently resistant to penicillin and ampicillin but sensitive to gentamycin, chloramphenicol, and tetracycline. 1.20 It has been postulated that the penicillin resistance is attributed to the production of beta-lactamase by the bacteria. Rapid resolution of the infection was obtained in our cases with topical and systemic gentamycin therapy.

The clinical picture of A. hydrophila corneal ulcer does not have specific morphologic characteristics that would permit diagnosis on clinical findings alone. The course is that of a hyperacute purulent exudation and corneal ulceration associated with hypopyon. History of contamination of the wound with water or soil from an aquatic source is significant. Rapid resolution of the ulcer follows antibiotic therapy directed toward gramnegative organisms.

SUMMARY

Two healthy men developed acute corneal ulcers caused by *Aeromonas hydrophila* after receiving traumatic foreign body injuries to the cornea. The ulcers resolved after treatment for gram-negative bacterial corneal ulceration. A history of contamination by water, soil, or material from an aquatic source was a significant factor in each case.

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EXCRETION OF GENTAMICIN IN RABBIT TEARS AFTER SUBCONJUNCTIVAL INJECTION

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Boston, Massachusetts

After subconjunctival injections, higher concentrations of gentamicin have been found in the cornea, sclera, choroidretina, and iris of normal rabbits' eyes than in inflamed (Staphylococcus aureus endophthalmitis) rabbits' eves. Two explanations seemed particularly likely: (1) inflammation increased ocular and orbital vascularity so that antibiotic was rapidly dissipated into the systemic circulation; or (2) needle puncture may have caused a larger rent in the inflamed than in the normal conjunctiva, permitting substantial escape of antibiotic into the tears. The first hypothesis is difficult to test because variations among animals in the volumes of distribution and rates of elimination · could easily mask differences in the kinetics of systemic absorption. Therefore, we examined the second possibility.

MATERIAL AND METHODS

We used eight New Zealand white rabbits weighing 1.4 to 1.8 kg (Table). In one animal, No. 1, both eyes were normal and only one was injected with antibiotic. In the remaining seven animals, we administered gentamicin into both eyes at the same time. In rabbits No. 2 and 3, the eyes were normal at the time of injection; rabbits No. 4 and 5 had unilateral endophthalmitis; and rabbits No. 6-8 had bilateral endophthalmitis. Ocular infection was produced by intravitreal injection of

approximately 75 colony-forming units of *S. aureus* 209P 48 to 72 hours before drug administration; the infection resulted in marked conjunctival inflammation and loss of red reflex.¹

To insure that all tears were collected, the nasolacrimal duct was blocked by application of cyanoacrylate² 24 hours before the injection of antibiotic. Complete obstruction was indicated by the failure of fluorescein dye to pass from the conjunctival sac into the nose.

Gentamicin (10 mg in 0.75 ml) was injected subconjunctivally (anterior subtenon's) at the 12 o'clock position, 3 to 4 mm from the corneoscleral limbus. Tears were collected over the next 90 minutes by placing 6.35-mm, filter-paper disks (of the kind used for antibiotic assay) in the cul-de-sac and replacing them when they were saturated. Disks obtained during each 15-minute interval were pooled and eluted overnight at 4°C in 1.0 ml of normal saline. Venous blood was drawn 5, 15, 60, and 90 minutes after the administration of antibiotic. Using Bacillus subtilis as the test organism,1 gentamicin concentrations in serum and eluted tear samples were assayed by the agardiffusion bioassay method.

RESULTS

The mean peak concentrations of antibiotic in tears were highest immediately after the injection, when they ranged from 5,000 to 7,000 µg/ml (Fig. 1). Thereafter, the levels declined steadily, but drug remained detectable even at 90 minutes. Although concentrations were slightly higher in the tears of animals with normal eyes, the differences were not statistically significant except at 30 minutes (P<.05).

From the Departments of Medicine, (Dr. Barza), and Ophthalmology (Ms. Kane and Dr. Baum), New England Medical Center Hospital and Tufts University School of Medicine. This study was supported in part by research grant EY01517 from the National Eye Institute.

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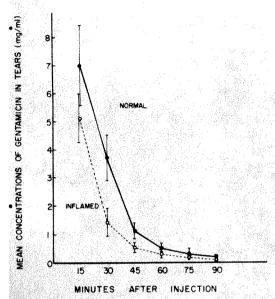


Fig. 1 (Barza, Kane, and Baum). Concentration of gentamicin in tears (mg/ml) at various intervals after subconjunctival injection in normal and inflamed rabbit eyes. Results shown are the mean and standard error of seven normal eyes and eight infected eyes.

Procedures done to one eye had no evident effect on the excretion of gentamicin in the tears of the fellow eye in the limited number of animals examined for this.

The major part of antibiotic excretion in the tears occurred during the first 30 minutes (Fig. 2). Overall, nearly 10% of the administered dose was retrieved in the

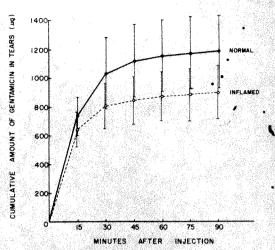


Fig. 2 (Barza, Kane, and Baum). Cumulative amount of gentamicin retrieved in tears (μg) at various intervals after subconjunctival injection in normal and inflamed (S. aureus endophthalmitis) rabbit eyes. Results shown are the mean and standard errors of seven normal eyes and eight infected eyes. There was no significant difference between the two groups at any interval.

lacrimal secretions. Again, differences between normal and inflamed eyes were not significant.

Peak serum concentrations were reached within 15 minutes after injection in all animals. There was no difference in peak concentrations between rabbits with both eyes normal and those with both eyes infected (Table). There was also no apparent difference among the animals in

TABLE

EXPERIMENTAL PROTOCOL AND PEAK SERUM CONCENTRATIONS

Rabbit No.	R.E.	L.E.	Peak Seru	m Concentration‡ (μg/ml)
1 2 3 3	s/c s/c s/c	s/e s/c s/c T s/c		28 32
5 6 7	s/c I, s/c I, s/c	I, s/c I, s/c I, s/c I, s/c		45 33

^{*}I designates infected (S. aureus endophthalmitis); s/c, gentamicin injected subconjunctivally. 1Shown only for animals in which both eyes were subjected to identical procedures.

the area under the serum concentration curves.

DISCUSSION

These data refute the possibility that after subconjunctival injection of gentamicin, the higher concentrations of the drug in the ocular tissues of normal as opposed to inflamed eyes are the result of different rates of drug loss in the tears. Indeed, excretion of gentamicin by this route was slightly (though not significantly) higher in normal eyes. The curves of serum concentrations showed no differences between animals with infected eyes and those with normal eyes; this shows that systemic absorption is rapid in both situations. The contribution of cross-diffusion of antibiotic from the fellow eye or from the systemic circulation can be ignored because previous experiments have shown only trivial concentrations (<3 to 4 μ g/ml) of gentamicin are detectable in the tears of the contralateral eye after injection of 20 mg of gentamicin subconjunctivally.3 Binding of gentamicin to proteins in the tears is of no consequence in this study because the antibiotic is only negligibly bound by serum proteins.4.5 Additionally, the tears were eluted in a fivefold or greater dilution of normal saline, which would render any binding inconsequential.6

SUMMARY

We subconjunctivally, injected gentamicin, in a dose of 10 mg, into normal and inflamed (Staphylococcus aureus endophthalmitis) rabbit eyes in which the nasolacrimal ducts had been blocked. We retrieved approximately 10% of the dose of drug in the tears, the majority in the first 30 minutes. The difference in lacrimal excretion of the drug between normal and inflamed eyes was not significant. Higher concentrations of gentamicinfound in the ocular tissues of normal eyes than in inflamed eyes cannot be attributed to loss in the tears.

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TOBRAMYCIN LEVELS IN HUMAN EYES

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Tobramycin (Nebcin) is a broadspectrum bactericidal antibiotic originally isolated from *Streptomyces teneb*rarius.¹ It is included in the class of aminoglycoside antibiotics together with gentamicin, kanamycin, neomycin, and others. The chemical structure of tobramycin is similar to that of gentamicin. It is stable and requires no refrigeration.

The antibacterial spectrum of this new compound includes Staphylococcus, Pseudomonas, Proteus, Escherichia coli, Klebsiella-Enterobacter group, and Serratia marcescens. Most of these organisms are major causes of serious ocular infections.

Tobramycin is not bound to serum protein. The drug is largely excreted unchanged in the urine by glomerular filtration. It is similar to gentamicin in many ways, but appears to offer the following advantages: (1) it produces less vestibulocochlear disfunction and less neuromuscular blocking activity; (2) it is more effective against *Pseudomonas* species²; and (3) it acts synergistically with carbenicillin (often successful in treating patients who had prior unsuccessful therapy with gentamicin or carbenicillin, or both).³

Tobramycin exhibits a marked prophylactic effect against *Staphylococcus* and *Pseudomonas* ulcers in rabbit eyes.⁴ Direct intravitreal injection of 500 µg causes no ocular toxicity.⁵

We designed the present study to determine the penetration of parenterally administered tobramycin into the blood and aqueous humor of human eyes subjected to routine intracapsular cataract extraction.

SUBJECTS AND METHODS

Written consent for the use of tobramycin was obtained from patients who were scheduled for routine intracapsular cataract extraction. (This study was performed before FDA approval of tobramycin for systemic use.) We excluded patients with a history of either otic or renal impairment. Tobramycin was administered either intramuscularly or intravenously at varying intervals before the. operation. Injections were given one-half, one, two, four, six, and eight hours preoperatively. Two patients per time interval received the injection. We obtained blood samples from all patients both preoperatively and at the time of aqueous humor removal for the time periods mentioned. After the corneoscleral limbus-based conjunctival flap and grooved incision at the corneoscleral limbus were performed, a 27-gauge needle attached to a dry, sterile, tuberculin syringe was used to remove a sample of aqueous humor. The aqueous humor was further drawn up into the syringe and the specimen was frozen for subsequent assay as described in a similar study.6 Blood samples were centrifuged and the serum separated for tobramycin assay. Samples were collected and shipped in dry ice, via air freight, to a laboratory for bioassay. The bioassay procedure used was the cylinder-place method with Bacillus subtilis.

There were 36 patients in the study, including 20 men and 16 women ranging in age from 45 to 79 years. No adverse effects were noted in the patients and no

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From the Department of Ophthalmology, University of Pennsylvania, and Scheie Eye Institute, Philadelphia, Pennsylvania.

TABLE 1
TOBRAMYCIN SERUM AND AQUEOUS
LEVELS FOLLOWING INTRAMUSCULAR
ADMINISTRATION OF 1 mg/kg

•	Aqueous Level (μg/ml)	Serum Level (µg/ml)
½ hour	0-0.08	1.84—3.73
_ ∤1 hour	0.12-0.12	3.60-5.00
2 hours	0-0.19	2.20-3.21
4 hours	0.22-0.32	2.24-2.90
6 hours	0.11-0.26	1.21-1.66
8 hours	0.12-0.22	0.93-2.31

changes attributed to the drug were reflected in their blood chemistry and urinalysis compared before and after treatment.

RESULTS

Recorded are the ranges of aqueous levels of tobramycin after an intramuscular dose of 1 mg/kg of body weight (Table 1). Ranges of aqueous levels after an intravenous dose of tobramycin (1 mg/kg of body weight, administered in 100 ml of normal saline over a one-hour period) were recorded as well (Table 2). Graphical representations of the value spread for intramuscular administration (Fig. 1) and intravenous administration (Fig. 2) are also shown.

Two patients who did not receive tobramycin served as controls and no antibiotic was detected in either their serum or aqueous humor.

TABLE 2
TOBRAMYCIN SERUM AND AQUEOUS
LEVELS FOLLOWING INTRAVENOUS
ADMINISTRATION 1 mg/kg

	Aqueous Level (μg/ml)	Serum Level (µg/ml)
¹ / ₂ hour	0-3.00	2.93—3.70
1 hour	3.00-4.00	2.38-2.52
2 hours	0.16-0.21	1.94-2.13
4 hours	0-0.10	1.66-1.66
6 hours	N/A	0.73-0.73
8 hours	N/A	0.65—0.79

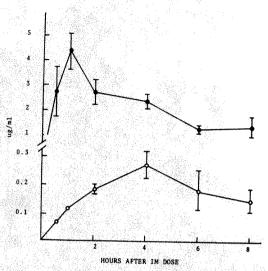


Fig. 1 (Furgiuele, Smith and Baron). Graphic representation of tobramycin serum and aqueous levels after intramuscular administration of I mg/kg of body weight. Open circles denote aqueous level; closed circles, serum level.

Two patients received intramuscular tobramycin in a dose of 3 mg/kg of body weight, one-half hour and one hour preoperatively. The aqueous humor levels were 0.20 μ g/ml and 0.34 μ g/ml, respectively. These levels approach the minimal inhibitory concentration for some isolates of *Staphylococcus* and *Pseudomonas*.

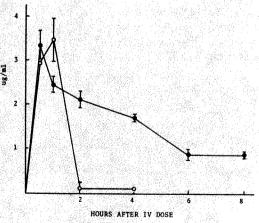


Fig. 2 (Furgiuele, Smith and Baron). Graphic representation of tobramycin serum and aqueous levels after intravenous administration of 1 mg/kg of body weight. Open circles denote aqueous level; closed circles, serum level.

Four patients received subconjunctival injections of tobramycin at varying time intervals before cataract surgery. Postoperatively, three patients received 20 mg of tobramycin subconjunctivally at 15 minutes, three hours, and $3^{1/2}$ hours respectively. The respective aqueous humor levels were $0.06~\mu g/ml$, $1.4~\mu g/ml$, and $1.0~\mu g/ml$. The patient who received tobramycin $3^{1/2}$ hours postoperatively demonstrated a vitreous level of $0.10~\mu g/ml$ (pars plana aspiration for vitreous sample). One patient received 10 mg of tobramycin $1^{1/2}$ hours preoperatively with an aqueous humor level of 4.8~mg/ml.

DISCUSSION

In cataract patients with a presumably normal blood ocular barrier, tobramycin is capable of penetrating into the aqueous humor. The levels detected are less than the minimal inhibitory concentration for the tested isolates of Staphylococcus, Pseudomonas, E. coli, and Proteus. However, these levels represent the degree of penetration after only a single parenteral dose equivalent to 1 mg/kg of body weight. If the recommended total daily dose of 3 mg/kg is given intramuscularly as a single injection, then the minimal inhibitory concentration is approached; and, for some of the bacterial isolates tested, the concentration is achieved.

One might expect bactericidal levels if the maximum total daily dose (5 mg/kg of body weight) was administered as a single injection. The molecular weight of tobramycin (467) is within the range of size expected to pass the blood aqueous barrier.7 Obviously, greater levels of the antibiotic would be likely to gain entrance under inflammatory ocular conditions when the blood ocular barriers are more permeable. Tobramycin administered subconjunctivally can achieve therapeutic aqueous humor levels that exceed the minimal inhibitory concentration of some isolates of Pseudomonas and Staphylococcus. The highest recorded aqueous

level occurred in the patient receiving 10 mg of tobramycin subconjunctivally $1^{1/2}$. hours preoperatively. Because we did not have a patient receiving 20 mg of tobramycin $1^{1/2}$ hours preoperatively, we have no way of comparing data. However, according to Baum and associates, the peak aqueous humor levels of gentamicin occur approximately one hour after subconjunctival injection.

SUMMARY

Tobramycin, a new aminoglycoside antibiotic, penetrated the aqueous humor of human eves after parenteral administration, either intramuscularly or intravenously, in 36 cataract patients with a presumably normal blood aqueous barrier. Tobramycin administered subconjunctivally to eyes with a normal blood aqueous barrier is capable of achieving therapeutic aqueous humor levels that. exceed the minimal inhibitory concentration of most isolates of Pseudomonas and Staphylococcus. No adverse effects were noted in the subjects studied. This drug may be used in the treatment of acute ocular infections.

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NOTES, CASES, INSTRUMENTS

COMBINED SCLERAL DEPRESSOR AND SCLERAL MARKER

HORST LAQUA, M.D.

Tübingen, West-Germany

For binocular indirect ophthalmoscopy and scleral depression I prefer a straight depressor, such as the Schocket indentor, or a simple cotton-tip applicator. However, it is bothersome to use a different instrument, usually a curved scleral marker on a thimble, to mark the hole during surgery. I therefore designed a straight instrument to be used alternately as a scleral marker and a scleral depressor.

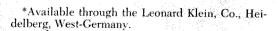
The instrument* is made of stainless steel and has a flat, corrugated handle for easy gripping. One end is formed in a ball-shaped fashion and serves as a depressor; the other end is cone-shaped. I drilled a hole into the cone and the rim was sharpened. Pressing the sharpened cone on the globe for a few seconds leaves a temporary mark on the sclera that can then be marked permanently with diathermy, cautery, or a pencil in the usual way. During surgery the instrument can be used alternately as a depressor and as a marker; it has served my needs well (Figure).

SUMMARY

I designed a combined scleral marker and scleral depressor for retinal detachment surgery.

From the Retina Service of the Univ.-Eye Clinic, Tübingen, West-Germany.

Reprint requests to Horst Laqua, M.D., Schleichstr. 12, Univ.-Augenklinik, 7400 Tübingen, West-Germany.



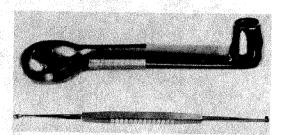


Figure 1 (Laqua). Combined scleral depressor and scleral marker. Top left, The ball-shaped end is used as depressor. Top right, The cone-shaped end serves as scleral marker. Total length of the instrument is 13.5 cm.

A NEW IRIS RETRACTOR

J.H.CH.HILGERS, M.D.

Curação, Netherlands Antilles

In cataract surgery, retracting the iris by some instrument is necessary before cryoextraction, unless a full iridectomy is done.

Such instruments range from cellulose sponge and forceps to metal or plastic iris retractors of various designs. They provide a wider pupillary opening for application of the cryoprobe and prevent iris adherence, thus making lens delivery easier.

It occured to me that a stainless steel wire would function similarly to an iris retractor, but with the following advantages: it would be more suitable to microsurgery, simple to maneuver, less traumatic to the iris, and easy malleability of the wire would facilitate retraction in patients with narrow pupils.

The iris-lasso* is made of a fine, but

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^{*}This instrument can be obtained from Leonhard Klein, Surgical Eye-Instruments, 6900 Heidelberg I, P. O. Box 103360, West Germany

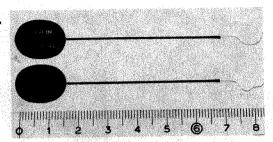


Fig. 1 (Hilgers). Iris-lasso with 50-mm shaft. Bottom, V-shaped wire. Top, semicircle wire. Eyelet at end of wire is open.

sturdy, stainless steel wire. It is connected to a shaft with a fingertip plate (Fig. 1).

The wire can easily be bent by a needleholder into any desired form, without losing its given form for normal use.

The length of the shaft may be varied on request. I started with a shaft of 50 mm with a fingertip plate at that distance. I now prefer a shaft of about 12 mm (Fig. 2).

Two forms for the curve of the wire are useful. One is a V-shaped form with a second bend in the upward leg. The bottom of the V facilitates introduction in a narrow pupil. To get in the second bend widens the pupil. This is done by maneuvering the fingertip plate.

The second form of the wire is a flattened semicircle for use in cases with more dilated pupils.

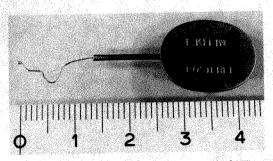


Fig. 2 (Hilgers). Iris-lasso with 12-mm shaft. Eyelet is closed.

If the wire is positioned under the iris, the surgeon can pull the iris-lasso to himself to widen the pupillary opening and the cryoprobe can be applied (Fig. 3).

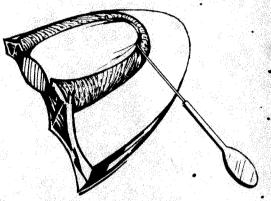


Fig. 3 (Hilgers). Stainless steel wire between shaft and eyelet is positioned under iris. By pulling fingertip plate to surgeon, pupillary opening is widened.

The wire may reach beyond the lens equator if the fingertip plate is not kept parallel to the iris-diaphragm; the vertical height of the V should be not more than 2 mm, the vertical height of the semicircular form not more than 1 mm.¹

Although the iris-lasso is intended for cataract surgery, it may also be useful in other procedures, such as removing intraocular foreign bodies and vitreous surgery.

SUMMARY

I successfully used the iris-lasso, a new iris retractor in the form of a stainless steel wire. The iris-lasso is simple to maneuver, suitable for microsurgery, and particularly useful in patients with small pupils.

REFERENCE

1. Nadler, M. P., and Pavlis, R. J.: Wider please! A study of pre-extraction pupillary dilatation. In Emery, J. M., and Paton, D. (eds.): Current Concepts in Cataract Surgery. St. Louis, C.V. Mosby, 1974, p. 70.

AMERICAN JOURNAL OF OPHTHALMOLOGY

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THE AMERICAN JOURNAL OF OPHTHALMOLOGY—SIXTY YEARS

This issue of THE JOURNAL marks the beginning of our sixty-first year of publication of the Third Series of THE AMERI-CAN JOURNAL OF OPHTHALMOLOGY. The First Series consisted of six issues published between 1862 and 1864 by Julius Homberger in New York. The Second Series was founded by Adolf Alt of St. Louis in 1884, a founder and first presi-'dent of the American Academy of Ophthalmology. The Third Series consolidated five journals; American Journal of Ophthalmology, Annals of Ophthalmology, Ophthalmic Record, Ophthalmology, and Ophthalmic Year Book and Literature. It brought the cost of these journals from \$34.50 annually to \$10 annually.

In many respects THE AMERICAN JOURNAL OF OPHTHALMOLOGY is unique in American medical publishing. It is the sole publication of the Ophthalmic Publishing Company, which is probably the

only remaining medical publishing company in America managed solely by physicians. It is not the official voice of any medical society, and is distributed by subscription only. It thus pays its own way and is not subsidized in any manner. It has no advertising agents and no subscription agents although it deals with many advertising and subscription agencies.

From its beginning THE JOURNAL has tried to encourage wide readership both in this country and abroad. Its list of 5,500 subscribers in countries outside the United States puts THE JOURNAL foreign list among the largest in the world. To encourage broad readership, THE JOURNAL has never charged a premium to libraries and other multiple-use subscribers. Indeed, considering the importance of our libraries to the furtherance of education and the health of our citizens it would seem counterproductive to seek such a premium. Foreign subscriptions are encouraged and not penalized with

high rates. Indeed, with the special handling and foreign postage required, it may be that foreign subscriptions provide less net income than domestic subscriptions. Conversely, The Journal has never had the staff to permit discount subscriptions to medical students and residents.

Since 1918 many technical innovations have improved THE JOURNAL although a page-by-page comparison indicates that the general format has remained the same. Now our printed page is arranged by photocomposition. THE JOURNAL is printed with giant offset presses that complete its print order of some 19,000 in the course of a few hours. The index is maintained by computerization. Its mailing list, as some chagrined subsc-*cribFers!-have discovered, is managed by a barely tamed computer.

No great changes are envisaged in the future. We plan a five-year index in the spring of 1978 and may publish a ten-year index in the spring of 1983. Special issues will be published from time to time as the occasion warrants. The Journal will continue its policy of having all papers reviewed by outside referees. To encourage candor and lack of bias in their reporting, these referees work anonymously. Authors too will continue to have the option of having their anonymity preserved during the review process. Thus far no one has taken this option.

Sixty consecutive years of publication by a group of interested physicians constitutes a milestone of sorts in American medical publishing. There are a number of reasons for this success. THE JOURNAL was not established as a profit-making venture and has generally hewn a close line between profit and loss. It has always had a group of loyal subscribers and supportive advertisers and it has never been necessary to provide costly campaigns to increase the numbers of subscribers or advertisers. As a publishing company with a single publication costs

are carefully controlled with attentive daily supervision of the operations. Authors have been outstandingly loyal; perhaps the single greatest problem facing THE JOURNAL is its inability to publish all of the meritorious material submitted. This is particularly true with the growth of ophthalmic research and its many skilled clinical observers in recent years. It is frustrating not to be able to publish interesting material, knowing that it will appear later in another publication.

THE JOURNAL and its Ophthalmic Publishing Company look forward to an interesting future. It seems likely there will be no new problems to face, the past having already provided a variety of experiences. The publishers though would most appreciate the recommendations of THE JOURNAL readers as it is for them that THE JOURNAL is published.

FRANK W. NEWELL

OBITUARY

Mrs. Audrey Hayden Gradle

Mrs. Audrey Hayden Gradle died October 13, 1977, in Santa Monica, California, after a long illness. She served as Execu-. tive Secretary of the Illinois Society for the Prevention of Blindness, as Director of the Missouri Commission for the Prevention of Blindness, and was active in founding the Southern California Society for the Prevention of Blindness, of which she was honorary president at the time of her death. In 1945 she received an Honorary Degree, Doctor of Law, from her alma mater, Oberlin College, for her contributions to preventing blindness. The St. Louis Society for the Blind gave her the Leslie Dana Medal in 1954, and the Pan American Association honored her at their meeting in San Juan in April 1974. Her husband, Harry S. Gradle, M.D., a prominent Chicago eye surgeon, was one of the founders of the Pan American

Association of Ophthalmology, and she served as a consultant.

The citation of her honorary degree in 1945 stated: "faith in scientific methods of treating the eyes, her extraordinary skill in winning cooperation of welfare agencies, school boards, and getting appropriations from state legislators to bring into being sight saving classes, eye testing projects, better lighting in school rooms. and clinics for the treatment of diseases of the eye." She was influential in Illinois in providing legislation to outlaw the sale of fireworks, to insure Crudé prophylaxis of the newborn, and establishment of special school classes for pupils with poor vision. She is survived by her sister and · eleven nieces and nephews.

BOOK REVIEWS

1977 Year Book of Ophthalmology. Edited by William F. Hughes. Chicago, Year Book Medical Publishers, 1977. Clothbound, 384 pages, table of contents, subject index, author index, 63 figures. \$22.75

The 15 sections of the Year Book embrace all phases of ophthalmology. Prefaced to each section is a perspective introduction by an acknowledged authority. The articles selected are from the world's major ophthalmic and related journals, and sagacious comment by Hughes is often added. Stress is given to the relative recent innovations such as vitrectomy, lens implants, ultrasonography, computed tomography (EMI scanning), photocoagulation, and axoplasmic transport. The reader frequently finds something worth remembering, such as the following examples:

Orbital mass lesions are accurately de-

termined by computed tomography and ultrasonography.

Cüppers faden operation consists in placing a suture of a rectus muscle into the sclera 12 to 14 mm posterior to its insertion. This reduces overacting muscle action and results in better coordinated eye movements.

In the adult, follicles at the corneoscleral limbus, semilunar fold, or caruncle suggest inclusion conjunctivitis. Tetracycline or erythromycin should be given orally four times daily for threeweeks.

In Mooren's ulcer the adjacent limbal conjunctiva contains collagenase and plasma cells. Excision of 4-mm width of this conjunctiva relieves pain and promotes healing.

Idoxuridine helps herpes simplex keratitis, but adversely affects herpes zoster lesions, but the reverse is true of topical corticosteroids.

The term "low-tension glaucoma" might well be abandoned since the field defects are determined by anemia, cardio-vascular disorders, and vascular hypertension.

A ten-year study showed that little value can be attached to the water provocative test as either a diagnostic or prognostic test in suspected open-angle glaucoma.

Automatic computerized perimetry is more exact and faster than conventional perimetry.

Tryptophan-deficiency cataract is the only authenticated cataract caused by a dietary deficiency.

For testing tonic pupil, pilocarpine 0.1% is an adequate substitute for Mecholyl, which is no longer marketed.

For recording visual fields at bedside, Amsler charts (1,2,3) can be used with a pinhead target.

A stenotic lesion in the common carotid artery can be detected better by carotid

compression tonography than by ophthalmodynamometry.

Secondary hemorrhage is less likely to follow traumatic hyphema if antifibrinolytic agents are given orally three times daily for five to six days—aminocaprioic acid (100 mg/kg of body weight) or tranexamic acid (25 mg/kg of body weight).

For an annual survey of the field of ophthalmology, the long established Year Book is paramount.

IAMES E. LEBENSOHN

Atlas of Strabismus Surgery, 2nd ed. By Eugene M. Helveston. St. Louis, C. V. Mosby Company, 1977. Clothbound, 262 pages, tables of contents, index, 382 black and white figures. \$35.50

The publication of the second edition of Atlas of Strabismus Surgery in just four years attests to its popularity. It also is an indicator of the rapid growth of pediatric ophthalmology and strabismology in the past decade.

This volume contains all the material of the first volume: the precise technique of various operations on each muscle, the clear illustrative line drawings and the concise accompanying text. Additionally, it contains a better description of Tenon's fascia. The picture of the orbit now depicts the fat that is so important to avoid in muscle surgery.

Several new features include the modern concept of the function of the superior oblique muscle with the anterior portion being mainly involved in torsion and the posterior in its vertical action. Attention is drawn to the second chance afforded by adjustable sutures where the amount of surgery has been grossly misjudged in the operating room. An entire chapter is devoted to the faden operation of Cüppers, also known as the posterior fixation procedure. The intriguing aspect of this operation is the need to increase

the innervation to an agonist to move the globe in one direction without giving a mechanical advantage to its direct antagonist. In Europe this procedure is most popular, whereas it is still being evaluated in this country.

My criticisms are minor. I dislike line drawings that may confuse the novice; for instance, on page 121 the superior oblique tendon looks like a rectus muscle, whereas in reality it may be a fairly gossamer structure. Secondly, I cannot follow the author's reason for listing the disinsertion of the inferior oblique muscle under Dyer's name while not mentioning Berke with the intrasheath tenotomy of the superior oblique muscle. I thing a more uniform policy of omitting all names but attaching reference numbers to the complete bibliography would be preferable. Then an interested reader could find and read the original article if he so desired.

I think this book is excellent and complete. All ophthalmologists interested in doing strabismus surgery can benefit from reading it.

PHILIP KNAPP

Atlas of the Ocular Fundus. Photographs of Typical Changes in Ocular and Systemic Disease, 2nd ed. By Hans Sautter, Wolfgang Straub, and Hermann RoBmann. Philadelphia, J.B., Lippincott Co., 1977. Clothbound, 160 pages, table of contents, index, 325 color figures, 2 black and white figures. \$49.00

Other atlases of the retina and the choroid are an antiquated method of teaching; most have limited discussions of the disease entities illustrated and reflect the bias of their authors in terms of abnormalities presented. By their very nature, retinal diseases have such an infinite variability that an atlas can not do justice to their many permutations. This atlas like-

wise suffers from these shortcomings—as well as deficiencies as the result of original publication in 1963 without obvious update for this English release.

There is no discussion of disease entities; stages in a disease process are not presented. This flaw is evident in the presentation of what is termed the "wet form of arteriosclerotic chorioretinopathy," a disorder known in this country as disciform macular degeneration. Although a series of 26 photographs are shown, there is no attempt to present them in terms of the pathogenesis of the disease. Far better to have somewhat fewer pictures with explanatory notes regarding the interrelated stages of this disorder. This is likewise true in the discussion of diabetes mellitus where no pictorial organization is attempted and no classification is provided.

There seems to be undue emphasis on certain topics such as 18 pictures of optic atrophy. The diseases noted next to the pictures do not begin to cover the spectrum of disorders that can cause this problem, yet the similarity of the photographs is obvious.

This atlas is the work of three outstanding German ophthalmologists; thus many terms used are not in vogue in this country and indeed probably are unknown to many of the younger ophthalmologists; for example, tabulated fundus and fundus flavus among others.

These disclaimers aside, most of the photographs are of good quality, and with the proper supervision, a younger ophthalmologist might profitably use this book in conjunction with other texts where the emphasis is more on the pathogenetic processes—seldom can one obtain 325 color figures for \$40. For the majority of ophthalmologists, however, this does not seem sufficient justification to warrant the purchase of this atlas.

RONALD E. CARR

Chirugie Plastique Orbito-Palpebrale. By J. Rougier, F. Hervouet, M. Lekieffre, P. Tessier, M. Woillez, and P. Derome. Paris, Masson, 1977. Paperbound, 498 pages, table of contents, index, 552 black and white figures.

The authors lament initially that more reconstructive ophthalmic plastic surgery is not performed by ophthalmologists. They suggest that the lack of general surgical training possibly explains this reluctance; a parallel situation is found in the plastic surgeon who is not trained in ophthalmology; collaboration is the answer. The authors of the monograph are three ophthalmologists, two plastic surgeons, and one neurosurgeon. Numerous collaborators are given credit in the introduction.

After a brief review of the surgical anatomy, methods of diagnosis, various surgical approaches to the orbit, orbital exenteration, evisceration, anophtholmos, and enucleation, the subsequent chapters deal with trauma and its sequelae. Of particular interest is the attempt to save vision after fractures involving the optic canal by decompression of the optic nerve. The authors state that the results obtained by the transethmoidal approach advocated by Japanese surgeons have not been duplicated.

The chapter on treatment of thyrotoxic exopthalmos is followed by an excellent review of Tessier's classification of craniofacial clefts, and by chapters describing the characteristics of many craniofacial anomalies and their treatment including craniofacial dyostosis and orbital hypertelorism. Of particular interest to craniofacial surgeons is Tessier's chapter on craniofacial surgery and the complications. His emphasis on restricting this type of surgery to centers where experienced members of a multidisciplinary team are trained to work together is the

States. The chapter contains an interesting discussion on a number of subjects including early surgical intervention, its influence on craniofacial growth, and contraindications to surgical intervention when the risks of morbidity and mortality are too great.

The chapter on blepharoptosis reviews briefly some of the operations described to shorten the levator aponeurosis. Surprisingly, frontalis suspension operations that are usually reserved for those patients who have no levator muscle function appear as alternatives to levator shortening operations. Among other chapters of particular interest are those devoted to blepharophimosis, ectropion, entropion, and trichiasis.

The chapter on reconstruction of the evelids surprises the American ophthalmic plastic surgeon, as the reader is advised never to use the upper eyelid for reconstruction of the lower eyelid by the tarsoconjunctival technique. Although it is agreed that the original technique advocated by Dupuy-Dutemps and Hughes caused deformity, entropion, and trichiasis in the upper eyelid, the tarsoconjunctival flap technique modified along the lines of the original Kollner technique has been highly successful and is of simpler execution than the chondromucosal graft combined with a rotation flap. The latter technique may have its indications in total destruction or ablation of the eyelid. The excellent technique of Mustardé for the reconstruction of the destroyed upper evelid by rotating the lower eyelid on a narrow vascular pedicle is described in some detail.

One of the last chapters deals with laophthalmos following facial paralysis. The authors advocate the Arion prosthesis or the use of magnets. In view of the complexity of the subject, the chapter entitled the "lacrimal apparatus" is brief.

The last chapter is devoted to medicolegal aspects, a timely subject.

The present trend toward the use of abbreviations of scientific terms such as DNA complicates reading a book written in French; the reader learns that DNA becomes ADN, FOF designates orbital facial clefts, DCF signifies craniofacial disjunction. These are minor points and do not detract from the considerable effort that was made to review plastic surgery of the orbit and its adnexa.

The book has great merit and is a noble effort to cover a wide variety of subjects.

Byron Smith John Converse

Ocular Therapeutics and Pharmacology, 5th ed. By Philip P. Ellis. St. Louis, C. V. Mosby Company, 1977. Clothbound, 283 pages, table of contents, index, 26 tables. \$19.50

In this fifth edition of Philip Ellis's textbook the aims and purposes of the first edition continue to be realized. The book serves as a quick reference for a busy practicing ophthalmologist, as a guide in therapy for beginning residents in ophthalmology, and for nonspecialists who plan to treat ocular disorders. Information in the book is presented in a concise form. As in the past the handbook is divided into two sections; the first deals with the basic considerations of treatment, and summarizes the present medical therapy of most ocular disorders. The second section presents the most commonly used medications that a practicing ophthalmologist would have to administer. Here one can find the actions, uses, side reactions, contraindications, preparations, and dosages of these agents. The dosages are always applied as those for adults but the methods for determining

pediatric doses are given at the beginning of the section. In this edition the pediatric dosage tables have been expanded and new therapeutic agents including antibiotics, anti-inflammatory drugs, enzyme inhibitors, autonomic nervous system agents, and anti-glaucoma medications have been added. New chapters have been added on carbonic anhydrase inhibitors and osmotherapeutic agents and on asesthetic agents.

Although each statement in the text is not referenced directly, an excellent refer-

ence list at the end of each chapter supplies a source of more detailed information to the ardent reader.

Dr. Ellis continues to provide us with an excellent, concise guide in the understanding and therapeutic management of patients with ocular disease. He has condensed his decades of considerable experience in pharmacological and therapeutic events to a brief, succinct, and lucid presentation.

IRVING H. LEOPOLD

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ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Albrecht von Graefes Archiv für Klinische und Experimentelle Ophthalmologie

PREVENTION OF CYSTOID MACULAR EDEMA AFTER LENS EXTRACTION BY TOPICAL INDOMETHACIN. Miyake, K. (Eye Hosp., Nagoya, Japan). Albrecht von Graefes Arch. Klin. Ophthalmol. 203:81, 1977.

Forty eyes undergoing intracapsular cataract extraction were treated with indomethacin as a 1% oil (sesame) solution. One drop was given the night before, three hours and one hour before surgery, and then three times a day for 40 days after surgery. This group was compared to 106 control eyes by using weekly fluorescein angiography to grade the extent of postoperative perifoveal retinal capillary leakage. The short-term results seven weeks after surgery show significantly less cystoid macular edema in the indomethacin-treated group than in the control group. Some degree of capillary leakage of fluorescein was seen in 33% of treated eyes versus 77% of control eves; severe leakage with decreased vision was found in none of the treated eyes but in 60% of control eyes. The author hypothesizes that prostaglandins form because of the trauma of surgery, diffuse through the vitreous and cause increased permeability of retinal capillaries. Indomethacin is known to inhibit prostaglandin synthesis. (2 figures, 1 table, 31 references)-Peter Egbert

American Journal of Diseases of Children

CAT EYE SYNDROME. Bofinger, M. K., and Soukup, S. W. (Children's Hosp. Res. Found., Cincinnati, Ohio). Am. J. Dis. Child 131:893, 1977.

The cat eye syndrome is characterized by colobomas, preauricular tags, and imperforate

anus as its most distinguishing clinical manifestations. This syndrome has been associated with an extra small acrocentric chromosome that after banding appears to be a 22 chromosome with partial deletion of the long arms. The infant reported here had narrow palpebral fissures and mild enophthalmos but no notable anomalies of the globe or fundi. There were preauricular skin tags and an imperforate anus. Cytogenetic studies showed that the child did in fact have a partial trisomy 22 but the mother showed a balanced translocation of chromosomes 11 and 22. (5 figures, 22 references)—Authors' abstract

American Journal of Medicine

THE OCULAR MANIFESTATIONS OF WEG-ENER'S GRANULOMATOSIS. Haynes, B.F., Fishman, M. L., Fauci, A. S., and Wolff, S. (National Inst. of Allergy and Infectious Diseases, Bethesda, Md.). Am. J. Med. 63:131, 1977.

Ocular manifestations of Wegener's granulomatosis may occur secondary to contiguous granulomatous sinusitis or as a result of focal vasculitis. Contiguous granulomatous sinus disease causes nasolacrimal duct obstruction, proptosis and ocular muscle or optic nerve involvement. Focal vasculitis unrelated to contiguous upper respiratory tract diseases is manifested by conjunctivitis, episcleritis, scleritis, corneoscleral ulceration, uveitis, and granulomatous vasculitis of the retina andoptic nerve. A review of 29 cases of Wegener's granulomatosis and three cases of lymphomatoid granulomatosis studied over the past 15 years at the National Institute of Allergy and Infectious Diseases disclosed single or multiple ocular manifestations of disease in 15 patients (47%). (8 figures, 3 tables, 91 references)—Authors abstract

Archives of Ophthalmology

THE CLINICAL SPECTRUM OF POSTERIOR POLYMORPHOUS DYSTROPHY. Cibis,

G. W., Krachmer, J. A., Phelps, C. D., and Weingeist, T. A. (Dept. Ophthalmol., Univ. of Iowa, Iowa City, Ia.). Arch. Ophthalmol. 95:1529, 1977.

The authors examined 61 affected members of eight families with an inherited corneal dystrophy. The corneal abnormalities varied greatly from one member of a family to another. Some patients had only a few isolated endothelial vesicles, while others in the same family had severe secondary stromal and epithelial edema. In some patients edema was present at birth or in early childhood; in others it developed later in life. The wide variation of corneal abnormalities suggests the possibility that several conditions previously described as separate disease entities, such as grouped vesicles, Schnyder's posterior herpes, posterior polymorphous dystrophy, and congenital hereditary endothelial dystrophy, are part of the clinical spectrum of expression of a single familial corneal dystrophy. The transmission in most of the families was autosomal dominant. In two families it appeared to be autosomal recessive. (34 figures, 20 references)-Authors' abstract

RETINAL ARTERIAL OCCLUSIVE DISEASE IN SYSTEMIC LUPUS ERYTHEMATOSUS. Gold, D., Feiner, L., and Henkind, P. (Dept. Ophthalmol., Montefiore Hosp., Bronx, N. Y.). Arch. Ophthalmol. 95: 1580, 1977.

Four patients with systemic lupus erythematosus (SLE) developed an unusual form of occlusive retinal arterial disease. The most prominent clinical features of this disorder were deposition of yellow-white material in retinal arterial walls and evidence of multifocal retinal arterial occlusion. Fluorescein angiographic findings included nonperfusion of the obstructed arteries and the retinal capillary beds fed by them, and fluorescein leakage at the sites of involvement of the retinal arteries. This ocular complication of SLE is presumably a manifestation of the widespread systemic vascular problems seen in this disorder. It may be more common in patients with lupus involving the central nervous system. (12 figures, 28 references)—Authors' abstract

OPHTHALMOPLEGIA AND ONDINE'S CURSE. Dooling, E. C., and Richardson,

E. P. (Massachusetts Gen. Hosp., Boston, Mass.). Arch. Ophthalmol. 95:1790, 1977.

The term Ondine's curse is used because of literary references to a similar state of suspended spontaneous respirations occurring in sleep when a mortal was cursed by the nymph Ondine. This is not peculiar to Leigh's disease but has also been observed in some newborn infants and with bilateral medullary infarcts, infiltrating brain stem glioma with involvement with the pontomedullary reticular formation, and the Pickwickian syndrome. (5 figures, 5 references)—David Shoch

ANTERIOR UVEITIS IN JUVENILE RHEU-MATOID ARTHRITIS. Kanski, J.J. (Canadian Red Cross Memorial Hosp., Berkshire, England). Arch. Ophthalmol. 95: 1794, 1977.

The ocular and systemic characteristics of 160 patients with anterior uveitis and seronegative juvenile rheumatoid arthritis are reviewed. Chronic uveitis occurred in 131 patients, 76% of whom were girls. Both eyes were involved in 70% of the cases. Band keratopathy occurred in 41% of the eyes, cataracts in 42%, and secondary glaucoma in 19%. Only 11 patients had uveitis before the onset of arthritis. Notable correlations included a pauciarticular onset of arthritis in 95% of the patients, and positive tests for antinuclear antibody in 82%. Of 29 patients with acute anterior uveitis, 27 were boys. The inflammation responded well to therapy and serious complications did not occur. At follow-up 21 patients had typical ankylosing spondylitis, and five had sacroiliitis. The incidence of positive results of tests for HLA-B27 antigen was 94%. (7 tables, 13 references)—Author's abstract

Aviation, Space and Environmental Medicine

HEAVY-ION-INDUCED CATARACTOGENESIS. Bonney, C. H., Hunter, D. M., Conley, G. E., and Hardy, K. A. (Radiation Sciences Div., USAF School of Aerospace Med., Brooks Air Force Base, Tex.). Aviation Space Environ. Med. 48:731, 1977.

Highly energetic nuclei are a characteristic of the galactic radiation spectrum. There have been very few studies on the effect of these heavy ions on the eye. Rhesus monkeys were exposed to stripped oxygen nuclei at energies of 250 MeV per nucleon. Ten months following exposure the animals began to exhibit changes in the posterior lens capsule of the irradiated eye. The opacities then progressed anteriorly following the pattern of cataracts induced by other forms of radiation. This pattern of changes at the plane of the posterior capsule seems to indicate a massive insult to the lens rather than to a more subtle localized insult to the equatorial terminal epithelium. In other studies the monkey lens has been found to respond very much like the human lens and therefore the results reported here are probably applicable to humans in galactic missions. (3 figures, 1 table, 20 references)—David Shoch

Developmental Medicine and Child Neurology

CONGENITAL HERPES SIMPLEX TYPE II INFECTION WITH EXTENSIVE HEPATIC CALCIFICATION, BONE LESIONS AND CATARACTS: COMPLETE POSTMORTEM EXAMINATION. Chalhub, E. G., Baenziger, J., Feigen, R. D., Middlekamp, J., N., and Shackelford, G. D. (Univ. Arkansas for Med. Sciences, Little Rock, Ark.). Devel. Med. Child. Neurol. 19: 527, 1977.

There have been many reports in the literature recently of neonatal herpes simplex and it has been assumed that these have been acquired in the perinatal period. These acquired herpes virus infections are mainly the result of infection by type II herpes simplex virus. Congenital herpes simplex virus infections are less common. This report documents the anomalies associated with this type of congenital infection. The infant reported here had the previously reported findings of microencephaly, microphthalmia, intracranial calcifications, chorioretinitis, congenital heart disease and abnormal digits. In addition this child also had extensive hepatic calcifications, osseous lesions, and cataracts. Rubella, cytomegaloviral disease, toxoplasmosis, hepatitis B and syphilis were excluded by serologic and viral studies. (7 figures, 1 table, 22 references)—David Shoch

Journal of the American Medical Association

WILMS' TUMOR METASTATIC TO THE ORBIT. Fratkin, J. D., Purcell, J. J., Krachmer, J. H., and Taylor, C. (Dept. Ophthalmol., Univ. of Iowa, Iowa City, Ia.). J.A.M.A. 238:1841, 1977.

In a 2½ old boy, a proptotic right lower lid developed one year after a primary abdominal mass proved to be Wilms' tumor. An orbital abscess or fungal infection was considered because the child was receiving chemotherapy. However, echography demonstrated a firm orbital mass, delineated its dimensions, and showed destruction of the orbital floor. The biopsy specimen showed metastatic tumor cells. Like neuroblastoma and certain hematologic and reticuloendothelial malignant neoplasms, Wilms' tumor may secondarily invade the ocular adnexa. (3 figures, 12 references)—Authors' abstract

Journal of Clinical Endocrinology and Metabolism

DYSTHYROID OPHTHALMOPATHY: ORBITAL EVALUATION WITH B-SCAN ULTRASONOGRAPHY. Forrester, J. V., Sutherland, G. R., and McDougall, I. R. (Dept. Ophthalmol. and Radiology, Southern Gen. Hosp., Glasgow, Scotland). J. Clin. Endocrinol. Metab. 45: 221, 1977.

B-scan ultrasonography was used to show muscle enlargement in all patients with Graves's disease and positive eye signs even when the clinical signs were minimal (lid lag and stare only). In addition, in cases of Graves's disease without clinical eye signs, 63% of orbital examinations showed ultrasonic evidence of muscle enlargement, often to a marked degree. This occurred more frequently in euthyroid patients after ablation of the thyroid by surgery or radiation, than in frankly thyrotoxic patients. The presence of subclinical ophthalmopathy in the fellow eye is a useful

diagnostic aid in cases of uniocular proptosis. (3 figures, 1 table, 14 references)—Authors' abstract

Klinische Monatsblaetter für Augenheilkunde

A NEW OPERATIVE METHOD FOR THE TREATMENT OF MALIGNANT GLAUCO-MA. Benedikt, O. (Univ. Eye Clinic, Graz, Austria). Klin. Monatsbl. Augenheilkd. 170:665, 1977.

Seven eyes with malignant glaucoma which were unresponsive to medical management were treated with cyclocryotherapy and all seven had good results. Eight to ten locations were frozen to -70° C for 60 seconds with a probe located so that the ice ball just reached the limbus. The applications were spaced 360 degrees around the limbus sparing the site of previous surgery. The intraocular pressure fell by the day after surgery and the anterior chamber deepened in one to five days. The author feels the drop in pressure was more than could be accounted for by decreased aqueous production. Therefore, he suggests cyclocryotherapy alters the cellular body or vitreous to permanently reverse the malignant glaucoma. (3 figures, 13 references)—Peter Egbert

RETINOPATHY IN THE COURSE OF A HOMOZYGOUS C HEMOGLOBINOPATHY. Babel, J., Regamey-Steiger, E., and Boreaux, G. (Univ. of Geneva, Geneva, Switzerland). Klin. Monatsbl. Augenheilkd. 170:804, 1977.

A 27-year-old black African female was noted, on a routine examination for mild iritis, to have typical sickle cell retinopathy. Hemoglobin electrophoresis revealed 98.6% HbC, indicating homozygous hemoglobin C disease (CC), instead of the expected SS or SC. Peripheral blood smear had many target cells but no sickle forms. Increased blood viscosity was measured. (4 figures, 11 references)—Peter Egbert

LIGHT INJURY TO THE RETINA. Henkes, H. E. (Erasmus Univ., Rotterdam, Netherlands). Klin Monatsbl. Augenheilkd. 170:813, 1977.

Light injury to the human eye has been

previously correlated only with well-known thermal effects (e.g., solar maculopathy, photocoagulation). One may speculate whether the light from strong conventional light sources may harm the retina. A case history is presented of a 21-year-old truck driver who was adjusting halogen headlights on his truck with both hands. The light came on and off several times, shining in his eyes, and he noted mild decrease in vision. Five days later, he needed to adjust the lights again and this time noted severe visual loss. Examination of his eyes showed bilateral dense central scotomas extending 15 degrees from fixation. The fundus showed large symmetrical gray-white macular lesions with irregular borders which blocked choroidal fluorescence during angiography. The lesions resembled those seen in acute posterior multifocal placoid pigment epitheliopathy. The patient's vision improved to normal over eight weeks. The macular lesions resolved to irregular pigmentary disturbances. Repeat examination five years later showed normal visual acuity, fields, electrooculography and electroretinography. (3 figures, 7 references)—Peter Egbert

A CHOROIDAL TUBERCULOMA SIMULAT-ING A MELANOMA. Blodi, F. C. (Univ. of Iowa, Iowa City, Ia.). Klin. Monatsbl. Augenheilkd. 170:845, 1977.

The clinical picture, the angiogram, the echogram, and especially the positive P 32 test led to the diagnosis of a choroidal melanoma in a 30-year-old man. Histologic examination revealed a tuberculoma. (5 figures, 2 tables, 1 reference)—Author's abstract

Neurology

OPHTHALMOPLEGIA AS A SIGN OF META-BOLIC DISEASE IN THE NEWBORN. Mac-Donald, J. T., and Sher, P. K. (Dept. Neurol., Univ. of Miami Med. Ctr., Miami, Fla.). Neurology 27:971, 1977.

Fluctuating ophthalmoplegia in the neonatal period is unusual. The authors report two infants, one with branched-chain keto-aminoaciduria, the other with nonketotic hyperglycinemia, who demonstrated varying degrees of ophthalmoplegia. In one, the abnormalities of the extraocular motility best correlated with elevations of serum leucine. In the other case, increased CSF glycine was

implicated. Varying ophthalmoplegia during the neonatal period should alert the clinician to consider an underlying metabolic disorder. (1 figure, 1 table, 17 references)—Authors' abstract

NEONATAL OPHTHALMOPLEGIA WITH MICROFIBERS: A REVERSIBLE MYOPATHY? Hanson, P. A., Mastrianni, A. F., and Post, L. (Dept. Neurol., Albany Med. College, Albany, N. Y.). Neurology 27:974, 1977.

An infant born with marked hypotonia showed prompt regression of skeletal muscle weakness, but by seven weeks of age had total external ophthalmoplegia. Biopsy of the gluteus muscle at 14 days showed marked variation in fiber size with a large proportion of very small fibers (less than 3µ). By ten months of age, biopsy of the vastus was virtually normal. The inferior oblique muscle was replaced by fibrous tissue containing a few remaining degenerating fibers. The child was normal at two years of age except for mild facial weakness and ophthalmoplegia. This syndrome may be the result of a reversible intrauterine process. (13 figures, 29 references)—Authors abstract

Science

A METHOD FOR DETECTING 8-METHOXYPSORALEN IN THE OCULAR LENS. Lerman, S., and Borkman, R. F. (Dept. Ophthalmol., Emory Univ., Atlanta, Ga.). Science 197:1287, 1977.

The psoralens are now being used in a variety of centers for the treatment of psoriasis. This treatment is usually associated with exposure of psoriatic lesions to ultraviolet light. There has been some discussion as to whether the drug might sensitize the lens to ultraviolet light and so facilitate the production of cataracts. Up to this time there has been no evidence that the drug itself does get into the lens. This paper indicates that following a intraperoneal injection of methoxypsoralen this material will appear in the lens in two and one half hours. The authors estimate that the concentration was about the order of about 10⁻⁵M. Thus at least the first requisite for ultraviolet radiation cataracts is present - that is the sensitized material

does get into the lens. (1 figure, 13 references)—David Shoch

Southern Medical Journal

EFFECT OF DELTA-9-TETRAHYDROCAN-NABINOL ON INTRAOCULAR PRESSURE, IN HUMANS. Cooler, P., and Gregg, J. M. (Depts. Ophthatmol. and Oral Surg., Univ. of North Carolina, Chapel Hill, N.C.). Southern Med. J. 70:951, 1977.

As early as 1971, it was noted that smoking marijuana lowered intraocular pressure. In this study one of the active components of marijuana, delta-9-tetrahydrocannabinol, was given intravenously to ten subjects with normal intraocular pressures. Two strengths were used - 0.022 mg/kg of body weight and 0.044 mg/kg of body weight. Intraocular pressure was found to decrease as much as 51% of baseline normal with an average decrease of 37%. Heart rate increased in a range of 22% to 65% of the resting pulse. Respiratory rate was not affected. No analgesic properties were demonstrated by either cutaneous or periosteal stimulation. Anxiety levels were increased by delta-9-tetrahydrocannabinol over placebo and diazepam (Valium). The mechanism of action is still uncertain but it is believed by some workers to be similar to that of a betaadrenergic stimulator, (6 figures, 2 tables, 9 references)-Authors' abstract

Transactions of the American Academy of Ophthalmology and Otolaryngology

PRIMARY OPTIC NERVE MENINGIOMAS: CLINICAL PRESENTATION AND MAN-AGEMENT. Wright, J. (Moorfields Eye Hosp., London). Trans. Am. Acad. Ophthalmol. Otolaryngol. 83:617, 1977.

Fifteen patients with meningiomas arising from the optic nerve sheath form the basis of this paper. Eleven patients were women; there was only one child. Thirteen patients had a similar clinical appearance. In each case visual loss was the first symptom. Proptosis, which occurred in only ten patients developed later (the time interval was three months to five years.) Optic disk edema or atrophy was pre-

sent in all 15 patients, some of whom had optociliary shunt vessels on the optic disk. The new fine-matrix EMI scanner (320 × 320) demonstrated the tumors but even clearer pictures of the lesion could be obtained using a c-mode ultrasonic scanner. The treatment of these meningiomas is essentially surgical. (5 figures, 1 table, 7 references)—Author's abstract

THE RELATIONSHIP OF EXTRAOCULAR MUSCLE PROBLEMS TO ORBITAL FLOOR FRACTURES: EARLY AND LATE MANAGEMENT. Helveston, E. (Dept. Ophthalmol., Indiana Univ. School of Med., Indianapolis, Ind.) Trans Am. Acad. Ophthalmol. Otolaryngol. 83:660, 1977.

Diplopia, in some or all fields of gaze, is the most common clinical finding in acute blowout fracture of the orbit. With restricted motility, it does not alone constitute a valid reason for early surgical intervention in blow-out fracture of the orbit. The majority of investigators believe that restricted motility after acute blow-out fracture is caused by soft tissue edema and hemorrhage, or from nerve damage to the inferior rectus, inferior oblique and medical rectus, or both rather than bony fixation of the muscles and fascia. Late motility problems are the result of fibrosis following edema, hemorrhage, or denervation. Late motility complications are as likely to occur in patients treated surgically as in those who are merely observed. Late motility problems after blow-out fracture of the orbit with or without repair are rare, difficult to treat, and similar to other types of restricted motility problems,

regardless of the cause. Thyroid myopathy, fibrosis after multiple surgeries, lost musclewith fibrosis, restricted motility after retinal detachment repair, and congenital fibrosis are some possible causes. Persistent ocular motility restriction with diplopia after orbital blowout fracture, with or without repair, should be treated by extraocular muscle surgery according to established principles. (5 references)—Author's abstract

THE ROLE OF CRYOSURGERY EXTERNAL OCULAR AND PERIOCULAR DISEASE. Fraunfelder, F. T., Wallace, T. R., Farris, H. E., Watkins, J., Hendrickson, R., Smead, W. J., and Limmer, B. L. (Dept. Ophthalmol., Univ. of Arkansas, Little Rock, Ark.). Trans. Am. Acad. Ophthalmol. Otolaryngol. 83:713, 1977.

Cryosurgery for basal or squamous cell carcinoma of the lid is easily performed, gives excellent cosmetic results, and has a low recurrence rate. It is not recommended for lesions involving the fornices, or sclerotic and morphea-type basal cell carcinomas, Tumor recurrences following radiation, surgery, or cryosurgery can still be retreated with cryosurgery. To date, there is no evidence that cryosurgery at temperatures above -40 C° causes damage to the lacrimal outflow system. Cryosurgery is of value in the management of trichiasis, reactive lymphoid hyperplasia, spider hemangioma, molluscum contagiosum, and conjunctival dysplasia. The 96% cure rate with one treatment for these lesions reported here is artifically high since the follow-up period is too short. (1 figure, 3 tables, 21 references)—David Shoch

NEWS ITEMS

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For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least three months before the date of occurrence.

International Evoked Potentials Symposium

The International Evoked Potentials Symposium
will be held in Nottingham, England, Sept. 4-6, 1978. Papers are invited from scientists and clinicians. For further information, write Colin Barber, Medical Physics Department, University Hospital, Nottingham NG7 2UH, United Kingdom.

AMERICAN ASSOCIATION OF PEDIATRIC OPHTHALMOLOGY: ANNUAL MEETING

The American Association of Pediatric Ophthalmology will hold its 1978 meeting July 5-8, 1978, in Williamsburg, Virginia. For additional information, write Keith W. McNeer, M.D., Suite 109, Grace Medical Village, 5700 West Grace St., Richmond, VA 23226.

GEORGETOWN UNIVERSITY: CENTER FOR SIGHT

Peter Y. Evans, professor and chairman of the Department of Ophthalmology, Georgetown University Medical Center, has announced the creation of the Center for Sight at Georgetown University. The Center for Sight, presently housed within existing facilities, will serve as a regional referral center for diagnostic and therapeutic ocular studies and research. The Center for Sight comprises various clinical subspecialty services, an ocular trauma unit, ophthalmic pathology laboratories, an ocular diagnostic unit, photography, clinical and basic research laboratories. A major fund-raising drive for construction of a new consolidated facility has been initiated. Michael Lemp, clinical associate professor, has been appointed clinical director of the Center for Sight.

University of Chicago: Annual Alumni Day

The University of Chicago Department of Ophthalmology will sponsor its Annual Alumni Day March 1, 1978, at the Albert Merritt Billings Hospital. Luncheon will be served before the scientific session. There are no fees and all ophthalmologists are invited to attend. For further information, write Ms. Danuta Sawicki, Department of Ophthalmology, 950 E. 59th St., Chicago, IL 60637; telephone (312) 947-6065.

University of Texas Medical Branch: 12th Annual Postgraduate Alumni Meeting

The University of Texas Medical Branch, Department of Ophthalmology 12th Annual Postgraduate Alumni Meeting will be held April 15, 1978, at the Clinical Science Auditorium, Galveston, Texas. The guest speaker will be William Havener. For further information, write Francis C. Skilling, M.D., Senior Resident, Department of Ophthalmology, University of Texas Medical Branch, Galveston, TX 77550.

GEORGETOWN UNIVERSITY CENTER FOR SIGHT: OPHTHALMIC TECHNICIAN PROGRAM

Georgetown University Center for Sight offers a two-year training program for ophthalmic technicians from June 1978 to June 1980. Two years of college, or an R.N., or L.P.N., or medical corps training, or equivalent, or certification as an opthalmic assistant is a required prerequisite. Qualified veterans will be given priority. Certification of successful completion of this approved program will qualify graduates to take the national certifying examinations for Ophthalmic Technicians by the JCAHPO. Application deadline is April 30, 1978. For further information, write Ms. Cadace P. Wolfe, Director, Ophthalmic Technician Training Program, Center for Sign, Georgetown University Medical Center, 3800 Reservoir Rd., N.W. Washington, DC 20007.

O'CONNOR HOSPITAL: PARS PLANA SURGERY WORKSHOP

An intensive workshop in microendosurgery of the vitreous, retina, lens, and iris via the pars plana will be held at O'Connor Hospital, San Jose, California, Jan. 27-28, 1978, and repeated Feb. 3 and 4. Enrollment is limited to 20 surgeons per course. Attention is directed almost exclusively to supervised practice. The program is designed for novice endosurgeons, those who would teach such surgery, and the ophthalmologist who wishes to pursue the question, "Is this for me and my hospital?" The workshop director is Conor O'Malley. The fee is \$400; residents \$225. For further information, write Mary Ann Miner, Workshop Coordinator, O'Connor Hospital, 2105 Forest Avenue, San Jose, CA 95128.

1977 HEED AWARD

Paul Lichter, M.D., received the Heed Award during the 1977 American Academy of Ophthalmology meeting in Dallas, Texas. The award was presented by Frank Newell, chairman of the Board of Directors of the Heed Foundation. Dr. Lichter received this award in recognition of his leadership and contributions in the field of ophthalmology. Dr. Lichter is associate professor of ophthalmology at the University of Michigan Medical School. The Society of Heed Fellows annually selects one of the Heed alumni for this award. Former recipients include Stuart Brown, Matthew Davis, Eugene Helveston, Philip Knapp, Harvey Lincoff, William Tasman, William Spencer, and Froncie A. Gutman.

GEORGETOWN UNIVERSITY CENTER FOR SIGHT: OPHTHALMIC ASSISTANTS COURSE •

Georgetown University Center for Sight will hold.

a ten-week ophthalmic assistants course June 26-Sept. 2, 1978. Sponsorship and employment by an ophthalmologist is a required prerequisite. Certificate of successful completion of this approved course plus one year of practical experience qualifies assistants for national certifying examination as an Ophthalmic Assistant-A by the Joint Commission on Allied Health Personnel in Ophthalmology. The tuition is \$200. The application deadline is April 30, 1978. For further information, write Ms. Candace P. Wolfe, Director, Ophthalmic Technician Training Program, Center for Sight, Georgetown University Medical Center, 3800 Reservoir Rd., N.W., Washington, DC 20007.

ROCHESTER OPHTHALMOLOGICAL • SOCIETY: 14TH ANNUAL ABRAM PINSKY LECTURE

The 14th annual Abram Pinsky Memorial Lecture sponsored by the Rochester Ophthalmological Soci-

ety will be delivered at The Marriott Inn, Rochester, New York, Feb. 9, 1978. Henry S. Metz professor of • ophthalmology and chairman of the Department of Ophthalmology of the University of Rochester, will be the featured speaker. His topic will be "Adjustable sutures in strabismus surgery."

UNIVERSITY OF UTAH: POSTGRADUATE SYMPOSIUM ON GLAUCOMA

The University of Utah will hold a postgraduate symposium on glaucoma March 3 and 4, 1978, at the Salt Lake Hilton Hotel, Salt Lake City. Registration fee is \$75 for practitioners and \$50 for residents. Faculty includes Paul Lichter, Steven Podos, Michael Kottler, and Henry Van Dyk, M.D. For further information, write Mrs. Jean Florence, Division of Ophthalmology, University of Utah Medical Center, Salt Lake City, UT 84132.

OREGON ACADEMY OF OPHTHALMOLOGY: 37TH ANNUAL POSTGRADUATER CONVENTION

The Oregon Academy of Ophthalmology will hold its 37th Annual Postgraduate Convention March 17 and 18, 1978, at the Sheraton-Portland, Portland, Oregon.

Guest speakers are Stuart I. Brown, Herbert E. Kaufman, Irving H. Leopold, and Ronald E. Smith. The local faculty will consist of staff members of the University of Oregon Medical Sciences Center and Devers Eye Clinic. Lectures will deal with infections and inflammatory disease of the lids and anterior segment.

Fee for members is \$100 if paid by March 8, 1978, and \$110 thereafter. For nonmembers the fee is \$125 if paid by March 8 and \$135 after March 8. This includes luncheon both days and the banquet. Residents are admitted without charge to the scientific sessions on presentation of a qualifying letter from their respective department heads. For further information, write Miss Rebecca Tarshis, Executive Secretary, 918 N.E. 44th Ave., Portland, OR 97213.

VISUAL FIELD CHARTS

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Papers are accepted on the condition that they have not been published or accepted for publication in any other journal, whether printed in English or any other language. On occasion, a paper read before a society and published in the society's transactions will be considered if the society publication does not reach as wide an audience as the contribution merits. When submitting such a paper,

the author must indicate the time and place of the meeting and the name of the society publication. It is not possible to coordinate the date of publication of such papers in THE JOURNAL with that in the society publication.

Authors will be advised promptly of receipt of their papers. Thereafter they will be advised within 30 days of acceptance, rejection, or need for revision. Manuscripts that require extensive editorial correction or retyping will be returned for that purpose.

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Fig. 1 (Jones, Smith, and Brown). Histologic section of the eye (hematoxylin and eosin, ×70).

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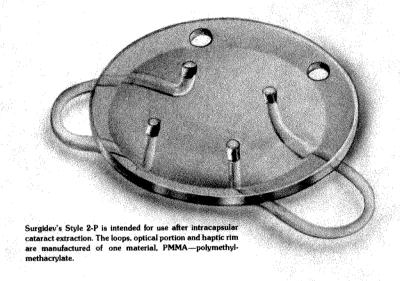
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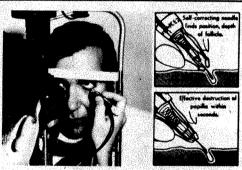
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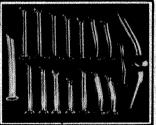
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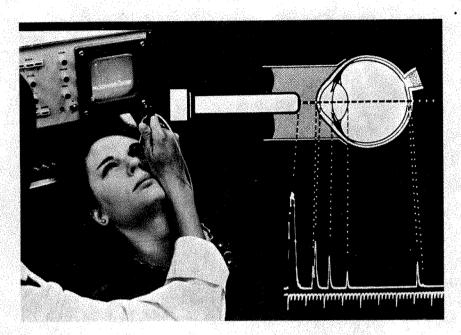
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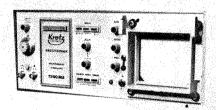
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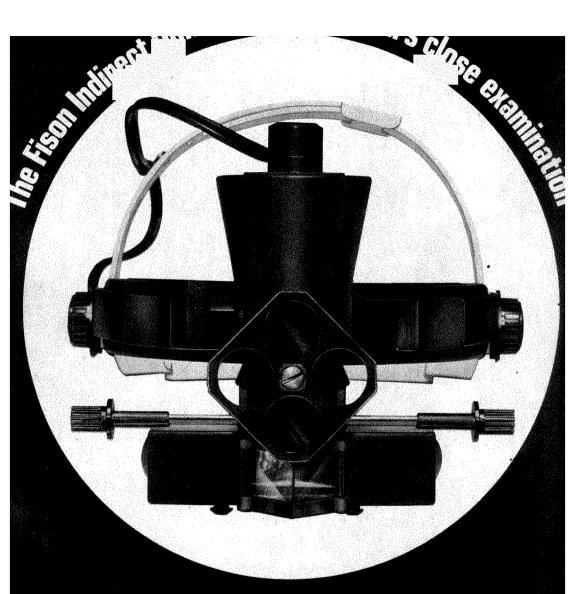
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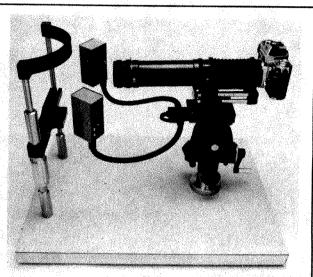
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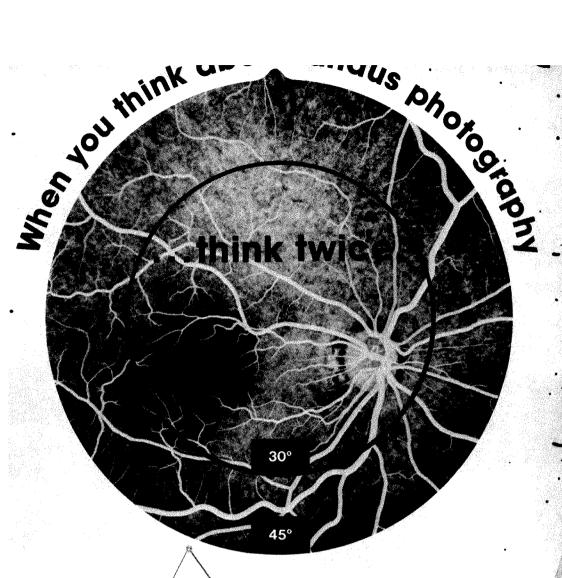
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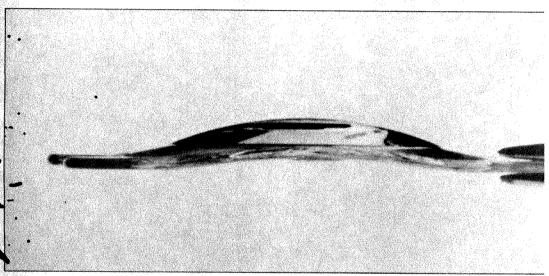
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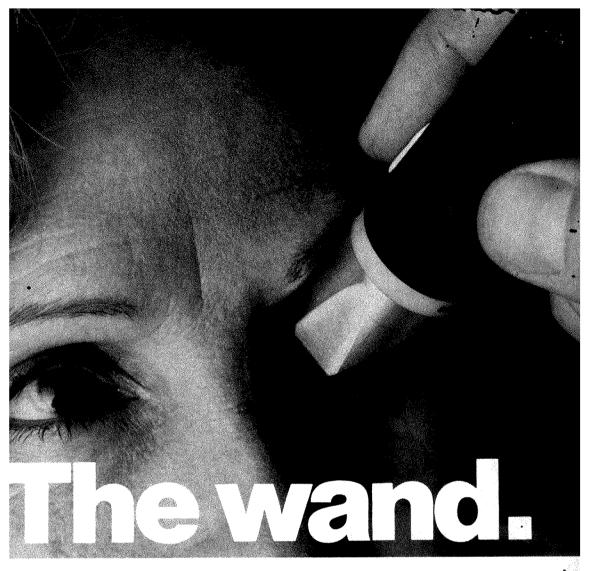
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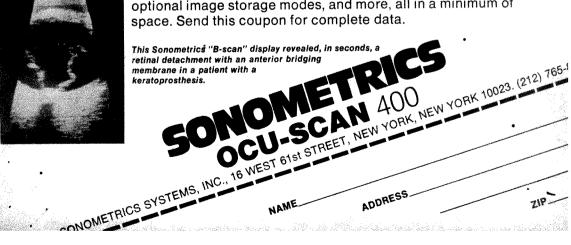
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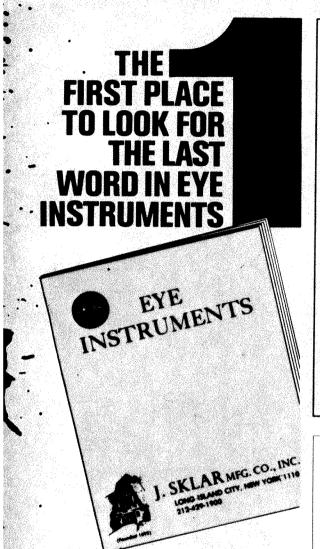
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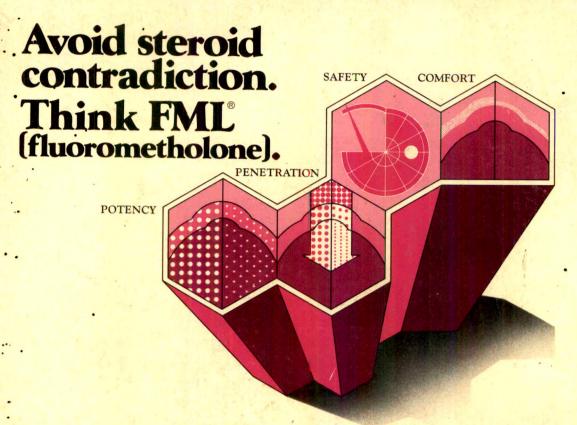




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REFERENCE NOTES:

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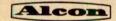
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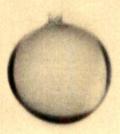
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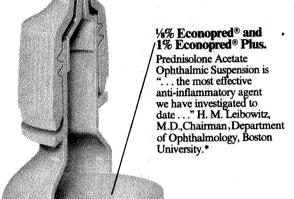


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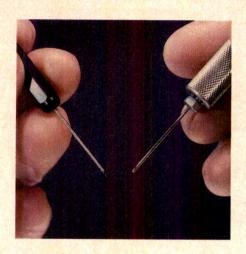
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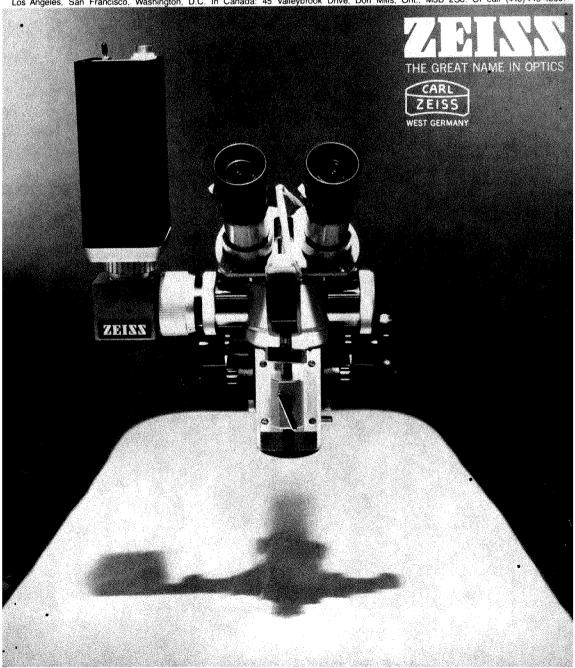
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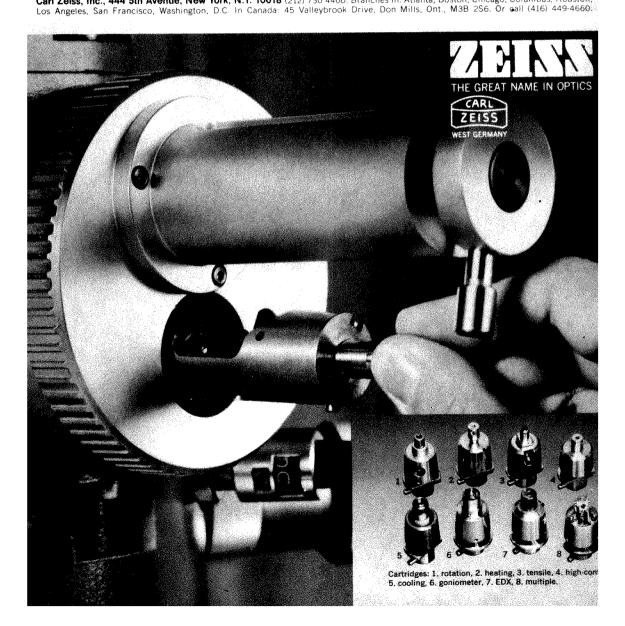
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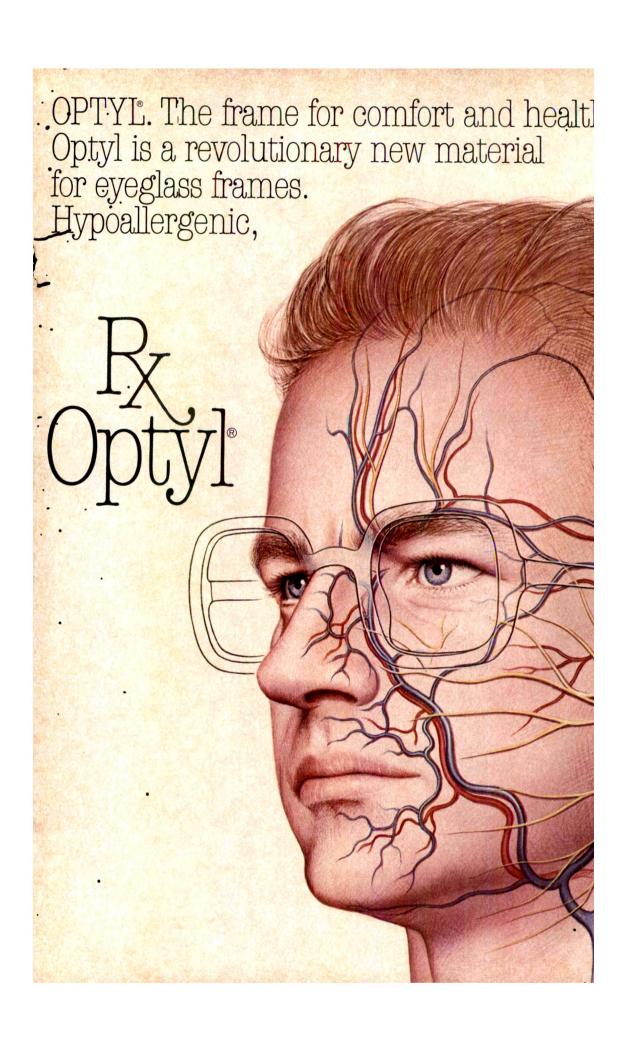
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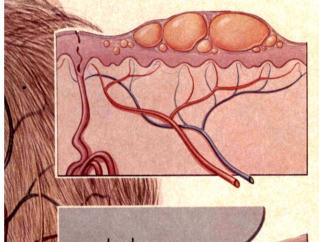
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CONTACT DERMATITIS...

Constant aposition of certain eyeglass frame materials may sensitize the skin and lead to contact dermatitis. In particular, cellulose acetate ester plastic (a common frame material) may not only produce irritants but also cause allergic reactions. With more inert materials there is less of a chance of a sensitivity reaction. And Optyl is almost completely inert biologically. In clinical studies of Optyl's allergenic potential, no skin irritation or contact dermatitis developed.

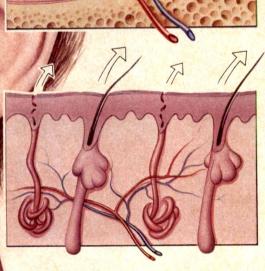
PRESSURE NECROSIS.

Continuous pressure from eyeglass frames may result in mild to severe pressure necrosis similar to decubitus ulcers ("bed sores"). The extent to which pressure necrosis may occur is in fluenced by the intensity and distribution of pressure and the duration of the exposure. Although the duration of exposure is usually limited by patient discomfort, severe point pressure of even short duration can damage cutaneous and underlying tissues. Optyl minimizes this threat, because of its light weight as compared to other common frame material.



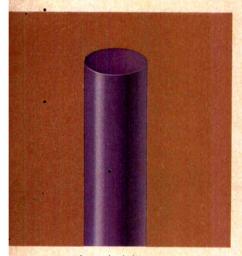
Eyeglass frames are subjected to a continual flow of facial glandular secretions. The sweat gland secretions are mostly water, with approximately 1% protein, lactate, uric acid, chloride and sodium electrolytes. The pH is slightly acidic. The sebum secreted by the sebaceous glands contains a large number of both common and unusual lipids, including triglycerides waxy esters, cholesterol, and squaline. Over a period of time, as these secretory products reac with cellulose acetate material, marked destruction occurs. However, because of its inert characteristics, Optyl minimizes reactions.

Reference: Jordan, W.P. Dahl, M.V.: Contact Dermatitis from Cellulose Ester Plastice. Archives of Dermatology 105:880, 1972



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Available monofilament, dyed.

New monofilament developed for ophthalmic procedures.

Smooth as only a monofilament can be:

In handling

In tissue

In tie-downs

—In addition to the other benefits VICRYL suture is known for. Available in 9-0 and 10-0 sizes on a wide variety of needles.

ETHICON the closing word



COMPLETE PRODUCT INFORMATION

VICRYL* (Polyglactin 910) Synthetic Absorbable Suture

DESCRIPTION VICRYL (polygiactin 910) synthetic absorbable suture is prepared from a copolymer of glycolide and lactide. These substances are derived respectively from glycolic and lactic acids. The empirical formula of the copolymer is (C₆H₂O₂)m(C₆H₄O₂)n.

VICRYL sutures are sterile, inert, nonantigenic, nonpyro-genic, and elicit only a mild tissue reaction during absorption. The braided and monofilament sutures are colored violet to enhance visibility in tissue. The braided suture is also avail-able undyed (natural).

ACTIONS Two important characteristics describe the *in* vivo behavior of absorbable sutures: first, tensile strength retention, and second, the absorption rate (loss of mass). Subcutaneous tissue implantation studies of VICRYL suture in rats show at two weeks post-implantation approximately 55% of its original tensile strength remains, while at three weeks approximately 20% of its original strength is retained. Intramuscular implantation studies in rats show that the absorption of VICRYL suture is minimal until about the 40th post-implantation day. Absorption is essentially complete between the 60th and 90th days.

INDICATIONS VICRYL synthetic absorbable suture is intended for use as an absorbable suture or ligature.

CONTRAINDICATIONS This suture, being absorbable, should not be used where extended approximation of tissues under stress is required.

WARNINGS The safety and effectiveness of VICRYL (polyglactin 910) suture in neural tissue, and in cardiovascular surgery have not been established.

Under certain circumstances, notably orthopedic procedures, immobilization by external support may be employed at the discretion of the surgeon.

Do not resterilize,

PRECAUTIONS VICRYL suture knots must be properly placed to be secure. Place the first throw in precise position for the final knot, using a double loop, tie the second throw square, using horizontal tension; additional throws are advisable.

Skin and conjunctival sutures remaining in place longer than 7 days may cause localized irritation and should be removed as indicated.

Acceptable surgical practice must be followed with respect to drainage and closure of infected wounds.

ADVERSE REACTIONS Reactions reported in clinical trials which may have been suture related have been mini-mal. These include skin redness and induration, rare in-stances of hemorrhage, anastomotic leakage, wound separation in the eye, and abscesses.

DOSAGE AND ADMINISTRATION Use as required per

HOW SUPPLIED VICRYL sutures are available sterile, as braided dyed (violet) and undyed (natural) strands in sizes 3 to 8-0, in a variety of lengths, with and without needles, and on LIGAPAK* ligating reels. VICRYL sutures, monofilament, dyed (violet) are available in sizes 9-0 and 10-0, in a variety of lengths with needles.

Also available in sizes 1 to 4-0 attached to CONTROL RELEASE* needles.

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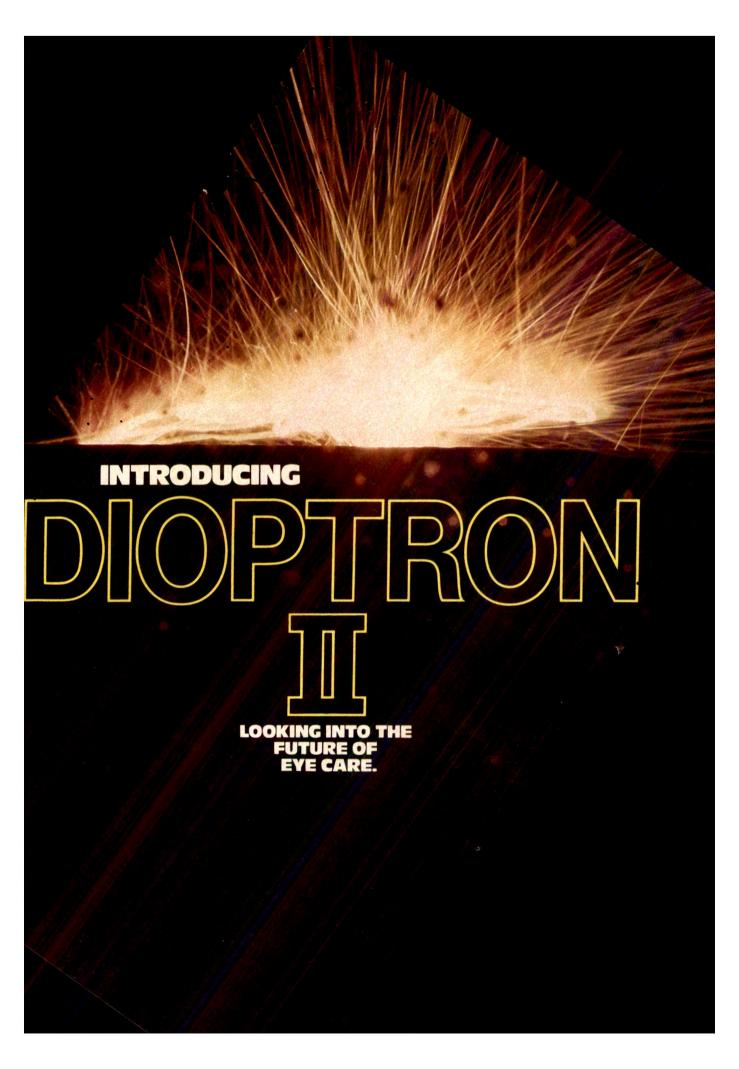
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At \$22,900, it was remarkable. Now at \$15,900, it's incredible. And money isn't the only thing you'll save!

Dioptron II
does everything you'd expect
from an autorefractor,
except it does it faster,
better and easier.

It saves time. Dioptron II cuts 2 to

It saves time. Dioptron II cuts 2 to 6 minutes off an average eye examination. And that will make your examinations faster, more efficient — and allow you to increase your patient load.

It saves trouble. Dioptron II makes it easy to handle your hard-to-handle patients. Since it requires no patient response, there's no patient confusion.

Dioptron II lets you handle your contact lens patients easier and faster than ever before. And after the lenses are made — use it to overrefract to verify a correct fit.

With aphakics, Dioptron II lets you simplify post-operative monitoring of astigmatism to help determine the final prescription.

Dioptron II is easy to use with children. They enjoy it. And since it doesn't require their response, you get faster, more objective information.

Dioptron II can give you results with even early cataract patients—a situation where retinoscopy is difficult, if not impossible.

And for your geriatric patients, Dioptron II means less decisions and examination fatigue for them — and faster, more accurate results for you.



It saves work. It's simple to use. Your receptionist can learn to operate it in 1 to 2 hours. So you're free to concentrate on the subjective examination.

It saves mistakes. Dioptron II gives you a more accurate starting point. Most autorefractors have a fixed

target that appears more blurred to some patients than others. Dioptron II has a "dynamic" target. That means it examines the patient's eyes, then "fogs" the target to the right degree. So every patient sees the target approximately the same way. You

get a perfect, objective starting point every time — without cycloplegics.

It saves you future problems. Dioptron II is 100% modular construction. It's built with its electronics in a pull-out drawer. Servicing is faster and easier. So when future improvements are made, you can simply add on a new module instead of replacing the entire instrument.

It's compact. Dioptron II is a single unit that needs only a 4 foot by 6 foot working space. It fits easily into any existing office layout.

It's adaptable. It works perfectly under normal office lighting. You can install it anywhere without creating special low-light conditions.

It's a Coherent product. You're getting the most technically advanced equipment available. With reliable, professional service behind it.

Coherent services

Coherent services start even before your Dioptron II arrives — with Practice Management Services. That means, at your request, we conduct a review of your office layout and procedures. We suggest where to install your Dioptron II. We help train your personnel. And we show

you how to introduce your patients to it. With Coherent, you get more than an autorefractor — you get a system with the most advanced before and after sale technical support.

DIOPTRON II SAVES YOU REAL MONEY. NOT JUST TAXES.

Equipment investments justified by tax write-offs are often marginal at best. But you don't have to consult your tax attorney to see that Dioptron II makes good economic sense.

With Dioptron II's new, affordable price — about half what you'd expect to pay — you'll come out ahead performing just 6 refractions a day. But more importantly, Dioptron II will allow you to increase your patient load dramatically.

IT PRACTICALLY PAYS FOR ITSELF.

If you added only one patient per day by using Dioptron II, in just one year it would pay for itself. And if yours is a high volume practice, you'll see results

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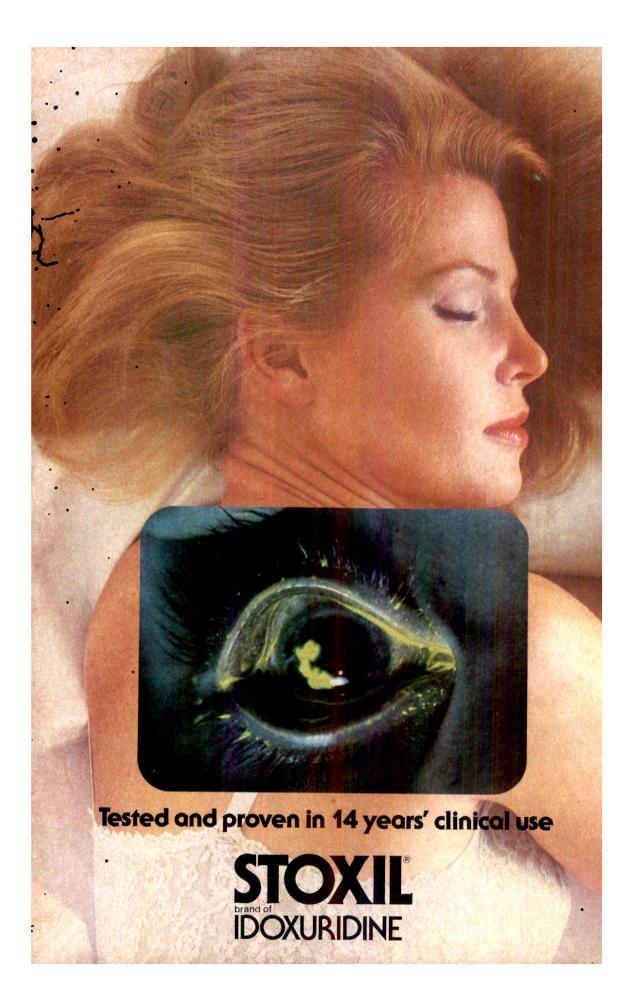
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The Standard for Herpes Simplex Keratitis

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Ointment 0.5% Solution 0.1%

- Never Exceeded in Clinical Effect
- Lower Cost than ara-A*
- Epithelial or Stromal Lesions

*Based on manufacturer's suggested list prices.

Check your local pharmacy for actual patient cost.

Before prescribing, see complete prescribing information in SKGF literature or PDR. The following is a brief summary.

Indication: Herpes simplex keratitis (topical use only).

Contraindications: Known or suspected hypersensitivity to any of the components.

Warning: Administer with caution in pregnancy or women of childbearing potential. Malformations were reported in one study in rabbits when idoxuridine was instilled in the eyes of the dams; a subsequent, more detailed study in rabbits showed no such effects, even at substantially higher dosages.

Precautions: If there is no response in epithelial infections after 7 or 8 days, other therapy should be considered. Recommended frequency and duration of administration should not be exceeded. Not effective in corneal inflammations if herpes simplex is not present. Boric acid should not be used concomitantly. To insure stability is maintained, the solution should not be mixed with other medications.

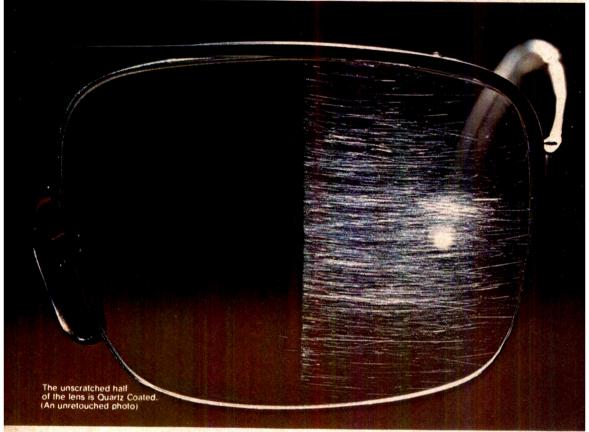
Adverse reactions: Occasionally, irritation, pain, pruritus, inflammation, or edema of the eye or lid; rarely, allergic reactions have been reported. Photophobia has occurred. Occasionally, small punctate defects (which may be a manifestation of the infection), corneal clouding, and stippling of the corneal epithelium have been observed.

Supplied: 0.1% Ophthalmic Solution (1 mg./ml.) in 15 ml. bottles with dropper; 0.5% Ophthalmic Ointment (5 mg./gram) in 4 gram tubes.

Smith Kline &French Laboratories Division of SmithKline Corporation Philadelphia, Pennsylvania



How did one side of this Hoya plastic lens get away with hardly a scratch?



If you were to rub the front surface of an ordinary resin lens a few times with scouring powder—and then do the same to the HOYA Hi-Quartz lens surface, you'd be amazed.

While the ordinary lens would be scratched, the Hi-Quartz would have come through the test virtually untouched. The secret is a quartz coating applied only to HOYA's own rigidly inspected lenses, giving them a high abrasion resistance. (Since over 95% of all scratches occur on the front surface of a lens, Hi-Quartz lenses are coated only on the convex side.) This means the lenses can take a lot of punishment.

Hi-Quartz also has a special multiple layer coating that increases light transmittance to nearly 96%, against the 92% of ordinary resin

lenses. So ghost images and reflections are reduced. HOYA resin lenses coated with Hi-Quartz also have durability, because the quartz and multiple coatings are firmly bonded together to the surface of the lens.

As an easy guide, the following types of resin lenses can be coated with Hi-Quartz: Single Vision; Flat Top 22, 25, 28; Round 22. These types of resin lenses cannot be coated with Hi-Quartz: Trifocal and Executive.

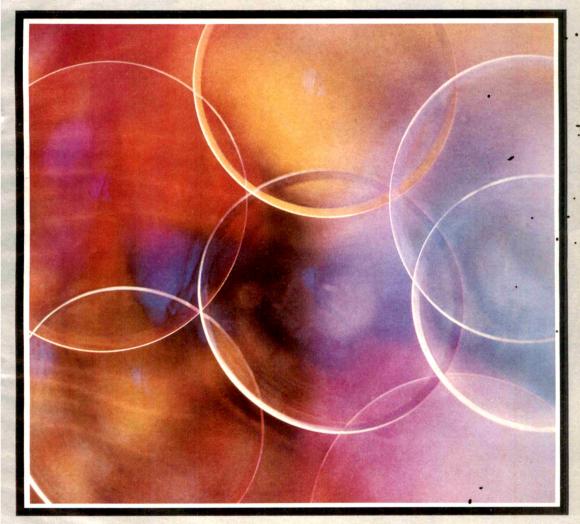
Finally, Hi-Quartz has all the advantages of ordinary resin lenses, including safety, lightness and ability to be dyed. (Dyeing must be done before the quartz coating takes place.)

HOYA's Hi-Quartz lens. When it comes to quality products HOYA hasn't even begun to scratch the surface.

*HOYA cannot accept cut or uncut lenses sent into the Torrance full prescription laboratory due to possible lens imperfections that could interfere with the quartz.

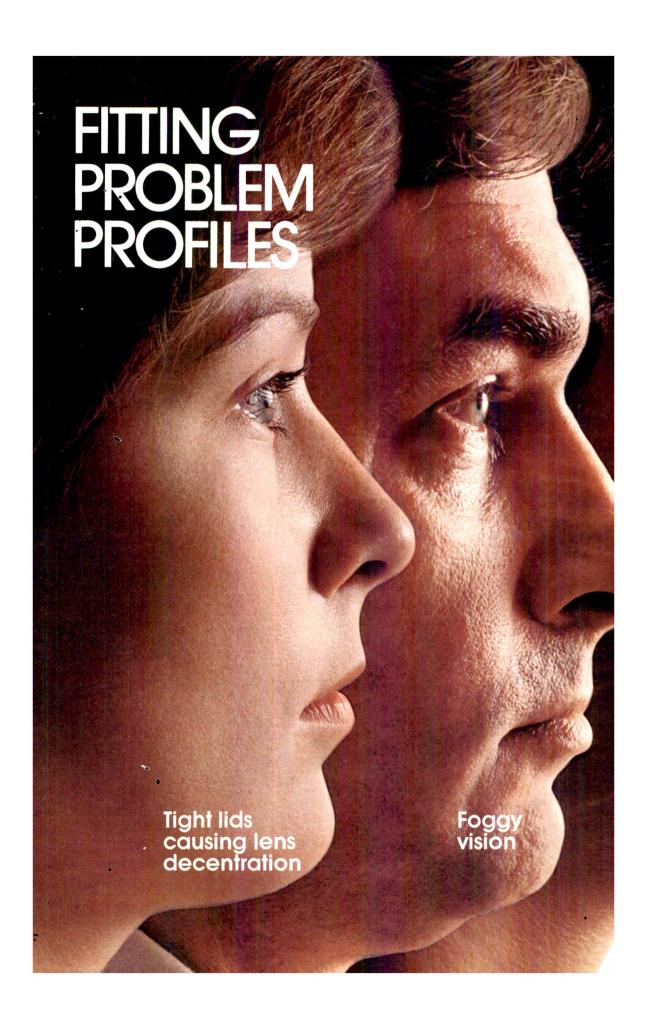
HOYA LENS OF AMERICA, INC.

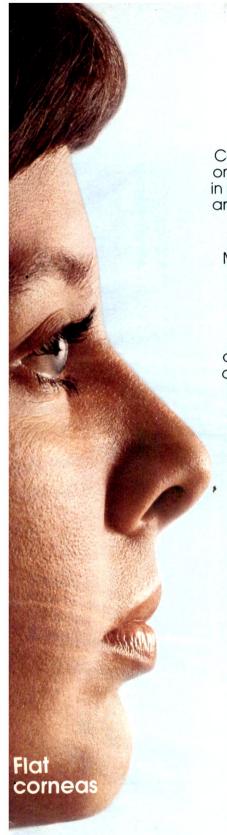
Bausch & Lomb introduces. UTRA-THIV U and U3 Series



SOFLENS® Contact Lenses (polymacon)

FOR "HARD-TO-FIT" PATIENTS





Ultra-Thin lenses are here to solve fitting problems for this special group of patients.

These thin-cast SOFLENS® (polymacon)
Contact Lenses have a "true" center thickness of
only .07mm. Available in 12.5 and 13.6 diameters
in powers from plano to —6.00D, Ultra-Thin lenses
are marked for easy identification—U for 12.5mm
diameter lenses and U3 for 13.6mm
diameter lenses.

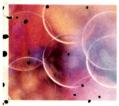
Many patients who have experienced foggy or variable vision may achieve "Best-Fit" with the thinner U and U3 Series. Patients who have not gained acceptable comfort before may experience less lens awareness and sensitivity with the Ultra-Thin Series. For patients who show decentration due to asymmetric or flat corneas or excessive influence by tight lids, the Ultra-Thin Series may provide "Best-Fit." And patients who have poor vision symptomatic of a steep fit may achieve "Best-Fit" with greatly improved visual acuity.

Made by Bausch & Lomb's spin-cast method,
Ultra-Thin lenses retain high reproducibility
and smooth optical surfaces. Fittings are
easy and efficient because the Ultra-Thin
Series lenses are fitted using the same simple
fitting system as with standard lens series.

To receive more information about Ultra-Thin Series of SOFLENS Contact Lenses, contact your sales representative or call, toll-free, 800-828-9030; in New York State, 800-462-1720.

Make your "hard-to-fit" patients good candidates for SOFLENS Contact Lenses with the Ultra-Thin Series.

BAUSCH & LOMB SOFLENS (polymacen) Contact Lenses





VISION CORRECTION USE

DESCRIPTION: The SOFLENS® (polymacon) Contact Lens is a hemispherical flexible shell which covers the cornea and may cover a portion of the adjacent sclera. It consists of 61.4% poly (2-hydroxethy) methacrylate) and 38.6% water by weight when immersed in normal saline. The material has a refractive index of 1.43 and the lens has a visible light transmittance of more than 27%.

ACTIONS: In its hydrated state a SOFLENS® (polymacon) Contact Lens is soft and pliable. When placed on the human cornea, the hydrated SOFLENS® (polymacon) Contact Lens acts as a refracting medium to compensate spherical ametropias.

INDICATIONS: SOFLENS® (polymacon) Contact Lenses are indicated for vision correction use in persons with non-diseased eyes and in aphakia.

CONTRĂINDICATIONS: SOFLENS® (polymacon) Contact Lenses are contraindicated in the epresence of any active untreated infection or abnormality of the anterior segment of the eye with the exception of ametropia and aphakia.

WARNINGS: Abrasions and Infections—If allens becomes less comfortable than it was when first placed on the wearer's cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present, or any abnormal eye condition is observed concurrent with lens wear, the lens should be removed immediately and a physician consulted.

Infectious corneal ulcers have been reported, usually associated with failure to follow the recommended procedures for care of the lenses

Aphakic Patients — Aphakic patients should not be fitted with SOFLENS® (polymacon) Contact Lenses during the postoperative period until, in the opinion of the surgeon, the eye has healed completely.

Lens Sanitation and Handling—Persons who require only vision correction and who would not, or could not, adhere to the recommended daily sanitary care of SOFLENS® (polymacon) Contact Lenses or who are unable to place and remove the lenses should not be provided with them. Failure to follow handling and sanitation instructions could lead to serious eye infections which might result in corneal ulcers.

Malfunction and rusting of the metal interior of the Aseptor®—Patient Unit as well as discoloration and cracking of the lens case has been reported after varying periods of use. If such occurs, appropriate replacement is indicated to avoid interference with the disinfection procedure.

Medicants and Eye Drops — When the lenses are used by persons requiring only vision correction no ophthalmic solutions or medicants, including conventional contact lens solutions and eye drops, should be used by SOFLENS® (polymacon) Contact Lens wearers prior to placement, or while the lens is in place, on the eye. Also, no solutions, including conventional contact lens solutions other than normal saline, and the solution made from the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets are to be used on a SOFLENS® (polymacon) Contact Lens when the lens is off the eye.

Wearing Restrictions—SOFLENS® (polymacon) Contact Lenses when used only for vision correction should be removed before sleeping or swimming and in the presence of noxious and irritating vapors. Insufficient lacrimal secretions or hypoesthesia may be restricting factors to the wearing of soft contact lenses

PRECAUTIONS: Storage—SOFLENS® (polymacon) Contact Lenses must be stored ONLY in normal saline solution. If left exposed to air, the lenses will dehydrate, become brittle, and break readily. If a lens dehydrates, it should be resoaked in normal saline solution until it returns to its soft, pliable state which may take as long as forty minutes.

Fresh normal saline must be prepared **daily** for cleaning and storing the lenses. The carrying case must be emptied and refilled with fresh normal saline solution just before disinfecting the lenses.

The one ounce squeeze bottle is intended for preparation of normal saline only. As this saline is not aseptic, it should not be used to clean a lens that is to be replaced immediately on the eye, nor should this solution ever be placed in the eye. If the patient wishes to remove a lens, clean it, or wet it and replace it on the eye, the solution in the carrying case should be used, as it will have been disinfected.

Hygiene — Hands must be washed, rinsed thoroughly, and dried with a lint-free towel before handling the lenses.

Cosmetics, lotions, soaps, and creams must not come in contact with the lenses since eye irritation may result. If hair spray is seed while the lenses are being worn, the eyes must be kept closed until the hair spray has settled.

Handling — A SOFLENS® (polymacon) Contact: Lens may be damaged by nicking or tearing if care is not exercised during placement on orremoval from the eye, replacing or removing it from the carrying case or in the cleaning process. Lenses must be placed very carefully in the carrying case to avoid damaging the edges of the lenses.

Disinfecting—Fresh normal saline must be prepared daily. After removal from the eye, the SOFLENS® (polymacon) Contact Lens must be irrigated with saline and rubbed gently to remove mucus and film from the lens "urface. The carrying case must be emptied and refilled with fresh normal saline solution just before disinfecting the lenses.

The causes and nature of deposits formed on the surfaces of some lenses have not been completely evaluated. However, some coatings are known to be proteinaceous and others may be oily or greasy film from extraneous agents, such as hair spray or other cosmetics, or from the patient's own lacrimal secretions. Many wearers experience little or no difficulty with such deposits. However, occasionally a wearer, who tends to secrete unusually large amounts of mudus in the lacrimal fluid, may experience a build-up of these deposits within a relatively few weeks, despite adequate cleaning measures. If surface accumulations of non-removable materials persist, professional care should be obtained promptly.

Deposits, characterized as medium or heavy, were found on 17.5 percent of a population of tenses worn an average of 11.2 months, including lenses worn as long as thirty months. The occurrence of these deposits appeared to increase with the duration of lens use. These

medium or heavy deposits, when they do exist, can be detected by means of a slit lamp biomicroscope examination. Light deposits, unrelated to length of lens use and of no apparent clinical significance, were observed on approximately one-half of the lenses studied.

The SOFLENS® Carrying Case should be washed at least once a week with hot water and then rinsed thoroughly with distilled water. Soap or other cleaners should never be used to clean the carrying case.

In order to remove protein deposits which may form on the lenses, wearers should use the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets according to the directions for use which accompany the tablets. To prevent the formation of the protein deposits, patients should use the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets once a week or as directed by the practitioner.

Disinfecting with an Aseptor® or Aseptron™ Disinfecting Unit is necessary to kill micro-

If an Aseptor® or Aseptron™ Unit is not available, the lenses may be disinfected by boiling them in their carrying case in a pan of water for 15 minutes. When this boiling method of disinfection is used, the lenses can be damaged if the boiling water is allowed to completely evaporate

rection is used, the lenses can be damaged if the boiling water is allowed to completely evaporate. The carrying case must **always** be tightly closed before disinfecting to prevent leakage of the saline from the case and subsequent dehydration of the lenses.

Fluorescein — Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with sterile normal saline solution and wait at least one hour before replacing the lenses. Earlier replacement may cause the lenses to absorb residual fluorescein.

ADVERSE REACTIONS: Serious corneal damage may result from wearing a SOFLENS® (polymacon) Contact Lens which has been soaked in a conventional contact lens solution. Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritation.

A lens adheres very rarely to an eye as a result of sleeping with the lens on, or wearing a hypotonic lens. If a lens adheres for any reason, apply normal saline and wait until the lens moves freely before removing it.

Clinical studies indicate that corneal edema, as manifested by symptoms such as rainbows, halos around lights, or foggy vision, may occur in less then 5% of SOFLENS® (polymacon) contact Lens wearers. If these symptoms occur, the lenses should be removed and professional consultation obtained.

Excessive tearing, unusual eye secretions, and photophobia are not normal. If these symptoms occur, the patient should be examined to determine their cause.

A faint blue haze, believed to be located in Descemet's membrane, has been reported in the

A faint blue haze, believed to be located in Descemet's membrane, has been reported in the Spokane, Washington area. As yet, the cause is unknown and the phenomenon has not been found elsewhere. The wearers report no subjective symptoms and there is no detectable effect on their visual acuity. There have been approximately 29 cases, and in 10 of these patients the blue haze has cleared or is in various stages of regression.

Neovascularization of the cornea has been observed in some aphakic patients fitted with the SOFLENS® (polymacon) Contact Lens, which may require discontinuation of the lens. Medical consultation should be obtained in such an instance.

LENS REPLACEMENT: Various studies have been conducted to determine the frequency of lens replacements and the reasons for those replacements. These studies show that during the first four months of wear, approximately one quarter of the lenses initially dispensed are replaced; the principal reason for these replacements is lens damage. Lenses are more apt to be damaged while new wearers are learning the prescribed handling and care techniques. After the initial four months of wear, the average lens replacement rate has been found to be approximately one lens per wearer per year. Lens loss, damage and surface deposits were the major causes for lens replacements.

DOSAGE AND ADMINISTRATION: Conventional methods of fitting contact lenses do not apply to SOFLENS® (polymacon) Contact Lenses. For a detailed description of the fitting technique, refer to the SOFLENS Fitting Guide, copies of which are available from: SOFLENS Division, Bausch & Lomb Incorporated, Rochester, N.Y. 14602

When the lenses are used only for vision correction, there may be a tendency for the patient to overwear the lenses initially. Therefore, the importance of adhering to the following initial daily wearing schedule should be stressed to these patients:

	Wear Time	Rest Period	Wear Time	Rest Period	Wear Time
Day	(hours)	(hours)	(hours)	(hours)	(hours)
1	3	1	3	1	3
2	3	1	3	1	3
3	4	1	4	1	4
4	4	1	4	1	4
5	6	1	6	1	4
6	6	1	6	1	4
7	8	1	8		
8	8	1	8		
9	8	1	8		
10	10	1	balance	of the waking h	ours*
11	12	1		of the waking h	
12	14	1		of the waking h	

*Lenses used only for vision correction should never be worn 24 hours a day.

When lenses are dispensed for vision correction, the wearer must be supplied with a lens care kit and must fully understand all lens care and handling instructions. As with any contact lens, regular recall visits are necessary to assure corneal health and wearer compliance with instructions.

HOW SUPPLIED: Each lens is supplied sterile in a glass vial containing sterile normal saline solution. The glass vial is marked with the manufacturing lot number of the lens and the dioptric power (black for plus power lenses; red for minus; gold or white for plano).

Caution: Federal Law Prohibits Dispensing Without a Prescription.

The SOFLENS* Care Kit is available for lens disinfecting, cleaning, and storage.

Complete information on lenses and accessory products can be found in the current SOFLENS Fitting Guide or price list.

DECEMBER 1976



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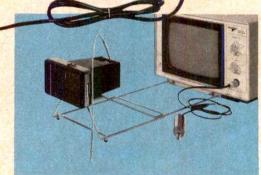
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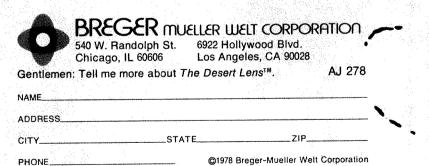
THE DESERT LENS. It solves an irritating problem nobody talks about-except patients.

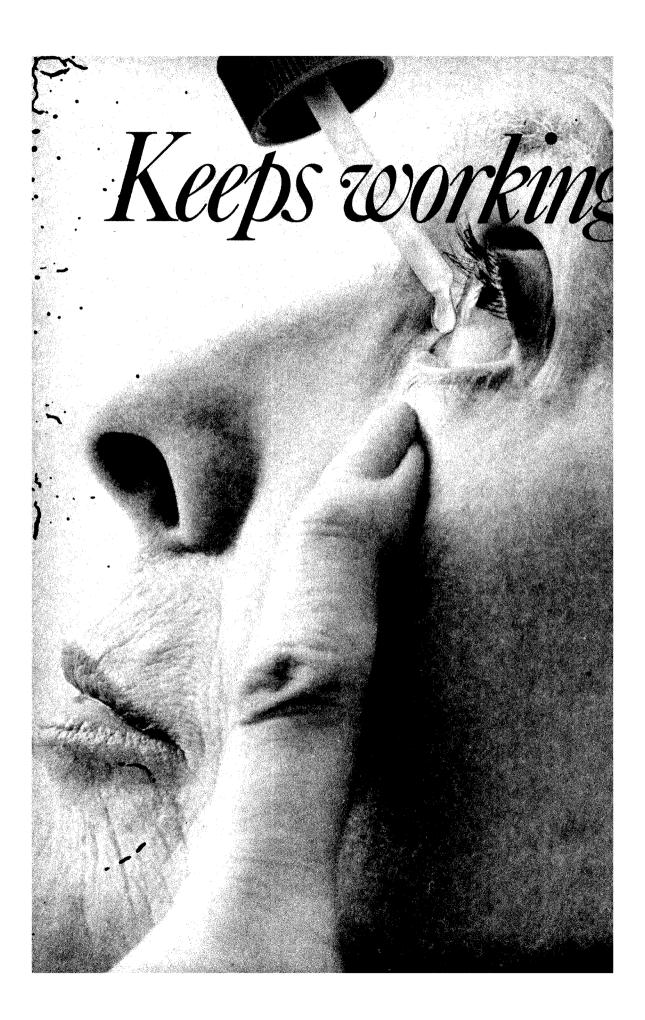
Low tear accumulation is a problem which causes discomfort for contact lens wearers. Especially those patients who live in the arid southwest; in dry, cold northern states; and wherever humidity is low.

Other lens manufacturers have failed to solve the problem. They've closed their eyes to it and waited hopefully for someone else to design a lens that encourages greater tear pooling. And now, somebody has. Us.

The Desert Lens™ (a special modification of the famous Flexinyl® thin, hard lens with flexure) is already giving the benefits of allday, trouble-free wear to patients who live in low-humidity climates and environments.

Like to know more? Mail the coupon and we'll send you the facts on The Desert Lens™. It will really open your eyes.





round the clock

One or two daily instillations – offer uninterrupted 24-hour control of IOP in:

□ chronic simple glaucoma
□ glaucoma secondary to aphakia

Minimize intraocular pressure peaks—whether they occur during the day or during critical early morning hours.

PHOSPHOLINE, IODIDE, is backed by two decades of clinical success as a longer-acting-not more potent-miotic.

In four available concentrations to satisfy individual patient • requirements.

□ Therapy with PHOSPHOLINE IODIDE should employ the lowest effective concentration. Therefore, the logical choice for initiation of therapy is the 0.03% strength—which probably has no greater potential for side effects than pilocarpine.

☐ Starting with this lowest concentration permits smooth transition to strengths of 0.06%, 0.125%, or 0.25% when required.

Note: After reconstitution, PHOSPHOLINE IODIDE remains stable for about one month at room temperature, or 12 months if refrigerated.

Phospholine Iodide — (echothiophate iodide for ophthalmic solution) ~

See next page for prescribing information



glaucoma secondary to aphakia

(Forfull prescribing information, see package circular)
PHOSPHOLINE IODIDE*

(ECHOTHIOPHATE IODIDE FOR OPHTHALMIC SOLUTION)

PHOSPHOLINE IODIDE is a long acting cholinesterase inhib-

itor for ropical use Indications: Glaucoma—Chronic open-angle glaucoma Subacute or chronic angle-closure glaucoma after indectomy or where surgery is refused or contraindicated. Certain non-uveitic secondary types of glaucoma, especially glaucoma following cataract surgery

Accommodative esotropia - Concomitant esotropias with a

significant accommodative component.

Contraindications: 1. Active eveal inflammation.

Most cases of angle-closure glaucoma, due to the possibility of increasing angle block.

 3. Hypersensitivity to the active or inactive ingredients Warnings: 1. Use in Pregnancy. Safe use of anticholinesterase medications during pregnancy has not been established, nor has the absence of adverse effects on the fetus or on the respiration of the neonate.

2 Succinylcholine should be administered only with great

2 Succinyicholine should be administered only with great caution, if at all, prior to or during general anesthesia to patients receiving anticholinesterase medication because of possible respiratory or cardiovascular collapse.

3 Caution should be observed in treating glaucoma with PHOSPHOLINE IODIDE in patients who are at the same time undergoing treatment with systemic anticholinesterase medications for myasthenia gravis, because of possible adverse additive effects.

Precautions: 1 Gonioscopy is recommended prior to initiation

of therapy.

2. Where there is a quiescent uveltis or a history of this condi-

Where there is a quiescent wents or a history of this condition, antich plinesterase therapy should be avoided or used cautiously because of the intense and persistent miosis and ciliary muscle contraction that may occur.

3. While systemic effects are infrequent, proper use of the drug requires digital compression of the hasolacrimal ducts for a minute or two following instillation to minimize drainage into the

nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation.

4. Temporary discontinuance of medication is necessary if salivation, urinary scontineance, diarrhea, profuse sweating, muscle weakness, respiratory difficulties, or cardiac irregularities.

5. Patier as receiving PHOSPHOLINE IODIDE who are ex 5 Patier as receiving PHOSPHOLINE (IODIDE who are ex-posed to carbamate or organophosphate type insecticides and pesticides (professional gardeners, farmers, workers in plants manufacturing or formulating such products, etc.) should be warned of the additive systemic effects possible from absorption of the pesticide through the respiratory tract or skin. During periods of exposure to such pesticides, the wearing of respiratory masks, and frequent washing and clothing changes may be advisable.

6 Anticholinesterase drugs should be used with extreme caution, if at all, in patients-with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulicer, pronounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects.

7. Anticholinesterase drugs should be employed prior to ophthalmic surgery only as a considered risk because of the possible occurrence of hyphema.

8. PHOSPHOLINE IODIDE (echothiophate lodide) should be used with great caution, if at all, where there is a prior history of retinal detachment.

Adverse Reactions: 1. Although the relationship, if any, of retinal detachment to the administration of PHOSPHOLINE IODIDE has not been established, retinal detachment has been reported in a few cases during the use of PHOSPHOLINE IODIDE in adult patients without a previous history of this disorder.

2. Stinging, burning, facrimation, lid muscle twitching, conjunctival and ciliary redness, browache, induced myopia with visual blurring may occur.

4. It is cysts may form, and if treatment is continued may enlarge and obscrure vision. This occurrence is more frequent in children. The cysts usually shrink upon discontinuance of the medication, reduction in strength of the drops or frequency of instillation. Parely, they may rupture or break free into the aqueous. Regular examinations are advisable when the drug is being prescribed for the treatment of accommodative esotropia.

5. Prolonged use may cause conjunctival thickening, obstruction of nasolacrimal canals.

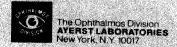
6. Lens opacities occurring in patients under treatment for glaucoma with PHOSPHOLINE IODIDE have been reported.

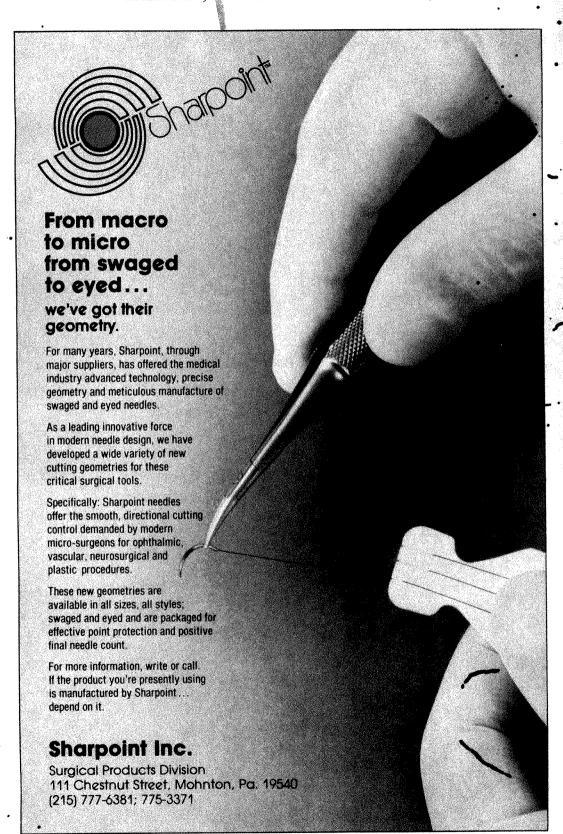
6. Lens opacities occurring in patients under treatment for glaucoma with PHOSPHOLINE IODIDE have been reported and similar changes have been produced experimentally in normal monkeys. Routine examinations should accompany clinical use of the drug.

clinical use of the drug.

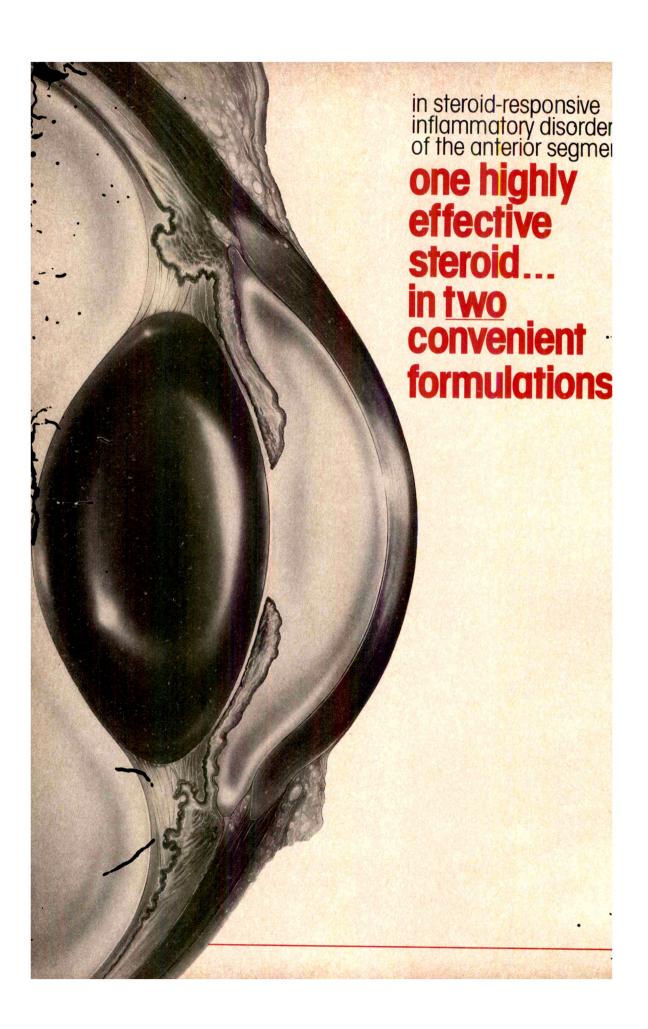
7. Paradoxical increase in infraocular pressure may follow anticholinesterase instillation. This may be alleviated by prescribing a sympathomimetic mydinatic such as phenylephrine.

Overdosage: Antidotes are atropine, 2 mg parenterally. PROTOPAM* CHLORIDE (pralidoxime chloride), 25 mg per kg intravenously; artificial respiration should be given if necessary. How Supplied: Four potencies are available. 1.5 mg package for dispensing 0.03% solution. 3.0 mg package for 0.06% solution, 6.25 mg package for 0.25% solution. Also contains potassium acetate (sodium hydroxide or acetic acid may have been incorporated to adjust pH during manufacturing), chlorobutanol (chloral derivative), mannitol, boric acid and exsiccated sodium phosphate.





. Maria de la composição de





Sterile Ophthalmic Solution

DECADRON Phosphate (DEXAMETHASONE SODIUM PHOSPHATE MSD)

0.1% Dexamethasone Phosphate Equivalent

- no eyedropper is necessary—the OCUMETER® ophthalmic dispenser conveniently dispenses one drop at a time
- remains stable at room temperature—may be carried in purse or pocket
- pH compatible with that of the eye



Sterile Ophthalmic Ointment

DECADRON® Phosphate . (DEXAMETHASONE SODIUM PHOSPHATE | MSD)

0.05% Dexamethasone Phosphate Equivalent

- melts below body temperature: medication spreads evenly over the entire eye preventing "blink-out"
- useful at bedtime or under a patch (to allow for individual patient needs)
- particularly useful when prolonged contact of steroid medication is indicated

Contraindications: Acute superficial herpes simplex keratitis.

Fungal diseases of ocular or auricular structures.

Vaccinia, varicella, and most other viral diseases of the cornea and conjunctiva.

Tuberculosis of the eye.

Hypersensitivity to a component of this medication.

Perforation of a drum membrane. Warnings: Employment of steroid medication in the treatment of stromal herpes simplex requires great caution; frequent slit-lamp microscopy is mandatory.

Prolonged use may result in glaucoma, damage to the optic nerve, defects in visual acuity and fields of vision, posterior subcapsular cataract formation, or may aid in the establishment of secondary ocular infections from pathogens liberated from ocular tissues.

In those diseases causing thinning of the cornea or sclera, perforation has been known to occur with the use of topical steroids.

Acute purulent untreated infection of the eye or ear may be masked or activity enhanced by the pres-

ence of steroid medication.

Usage in Pregnancy—Safety of intensive or protracted use of topical steroids during pregnancy has not been substantiated.

Precautions: As fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid applications, fungus invasion must be considered in any persistent corneal ulceration where a steroid has been used or is in use.

Intraocular pressure should be checked frequently.

Adverse Reactions: Glaucoma with optic nerve damage, visual acuity and field defects, posterior subcapsular cataract formation, secondary ocular infection from pathogens including herpes simplex liberated from ocular tissues, perforation of the globe.

Rarely, filtering blebs have been reported when topical steroids have been used following cataract surgery.

Viral and fungal infections of the cornea may be exacerbated by the application of steroids.

Rarely, stinging or burning may

occur.

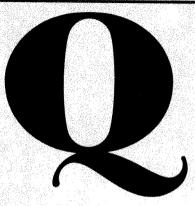
How Supplied: Sterile ophthalmic solution in 2.5-ml and 5-ml glass bottles with dropper assembly and in 5-ml OCUMETER® ophthalmic dispensers, containing per milliliter of buffered solution: dexamethasone sodium phosphate equivalent to 1 mg (0.1%) dexamethasone phosphate; creatinine, sodium citrate, sodium borate, polysorbate 80, disodium edetate in the OCUMETER, sodium hydroxide to adjust pH in glass bottles, hydrochloric acid to adjust pH in plastic dispensers, water for injection, and sodium bisulfite, phenylethanol, and benzalkonium chloride added as preservatives. Ophthalmic ointment in 3.5-g tubes, containing per gram: dexamethasone sodium phosphate equivalent to 0.5 mg (0.05%) dexamethasone phosphate; white petrolatum and mineral oil.

For more obtailed information, consult your MSD representative or see full prescribing information.

Merck Sharp &

Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, Pa. 19486





Which is the only meeting in 1978 you cannot afford to miss?

... if you do Cataract Surgery.

... if you have any interest in IOL's.

...if you are experienced in IOL's.

...if you appreciate the luxury of the CPH.

...if you know how the AIOIS puts on a meeting.

Approved for AMA Continuing Medical Education credit, category I, for 40 hrs.



WHEN: MARCH 14-18, 1978

WHERE: CENTURY PLAZA HOTEL, LOS ANGELES, CALIFORNIA

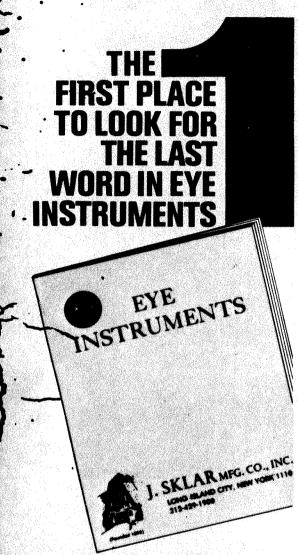
WHY: FOUR DAYS DEVOTED EXCLUSIVELY TO IOL'S

Methods: Exhibits, Lectures, Panels, Group Sessions, Video Presentations on Hotel Room TV's, Symposia

Topics: All aspects of IOL Science: Techniques Old and New, Iris-support Lenses, Anterior and Posterior Chamber Lenses, Endothelial Results, CME Studies, Vitrectomy, Results, Complications and Treatment, New Lenses and Instruments

Speakers: Every major name on the subject of IOL's

Prior to Feb. 1: Member	COST s of AIOIS \$250 ☐ I Nurses and Technic		☐ Spouses \$50
After Feb. 1:	of AIOIS \$300 \[\[\sum \] Nurses and Techr		☐ Spouses \$75
Cancellations: 90% refund prior payable to: U.S. Intraocular Len California 90403. For hotel Avenue of the Stars, Los Angele	s Symposium and send wit reservations, please o	h this form to: P.O. Box ontact Century Plaz	3140, Santa Monica, a Hotel directly at
payable to: U.S. Intraocular Len California 90403. For hotel	s Symposium and send wit reservations, please o	h this form to: P.O. Box ontact Century Plaz	3140, Santa Monica, a Hotel directly at
payable to: U.S. Intraocular Len California 90403. For hotel Avenue of the Stars, Los Angelo	s Symposium and send wit reservations, please o	h this form to: P.O. Box ontact Century Plaz	3140, Santa Monica, a Hotel directly at
payable to: U.S. Intraocular Len California 90403. For hotel Avenue of the Stars, Los Angelo Name	s Symposium and send wit reservations, please o	h this form to: P.O. Box ontact Century Plaz	3140, Santa Monica, a Hotel directly at



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GARAMYCIN

gentamicin sulfate, U.S.P.

OPHTHALMIC

Solution-Sterile Ointment-Sterile

Each mt. or gram contains gentamicin sulfate equivalent to 3.0 mg gentamicin.

DESCRIPTION Gentamicin sulfate is a water-soluble antibi-

DESCRIPTION Gentamicin sulfate is a water-soluble antibi-otic of the aminoglycoside group active against a wide variety of pathogenic gram-negative and gram-positive bacteria GARAMYCIN Ophthalmic Solution is a sterile, aqueous solution buffered to approximately pH 7 for use in the eye Each ml. contains gentamicin sulfate (equivalent to 3.0 mg, gentamicin), disodium phosphate, monosodium phosphate, sodium chloride, and benzalkonium chloride as a preserva-tive

gentamicini) disolitin prospirate, monosculum prospirate, sodium chloride, and benzalkonium chloride as a preservative.

GARAMYCIN Ophthalmic Ointment is a sterile ointment, each gram containing gentamicin sulfate (equivalent to 3.0 mg. gentamicin) in a bland base of white petrolatum, with methylparaben and propylparaben as preservatives.

ACTIONS The gram-positive bacteria against which gentamicin sulfate is active include coagulase-positive and coagulase-negative staphylococci, including certain strains that are resistant to penicillin; Group A beta-hemolytic and non-hemolytic streptococci; and Diplococcus pneumonae. The gram-negative bacteria against which gentamicin sulfate is active include certain strains of Pseudomonas aeruginosa, indole-positive and indole-negative Proteus species. Schenicha coli. Klebsiella pneumonae (Friedlander's bacillus). Haemophilus anfluenzae and Haemophilus aegyptius (Koch-Weeks bacillus). Aerobacter aerogenes. Moraxella lacunata (diplobacillus of Morax-Axenteld), and Neisseria species, including Neisseria gonorrhoeae. Although significant resistant organisms have not been isolated from patients treated with gentamicin at the present time, this may occur in the tuture as resistance has been produced with difficulty in vitro by repeated exposures.

INDICATIONS GARAMYCIN Ophthalmic Solution and Ointment are indicated in the topical treatment of infections of the external eye and its adnexa caused by susceptible bacteria. Such infections embrace conjunctivitis, keratitis and keratoconjunctivitis, corneal ulcers, blepharitis and blepharoconjunctivitis, corneal ulcers, blepharitis and blepharoconjunctivitis.

CONTRAINDICATIONS GARAMYCIN Opnthalmic Solution and Ointment are contraindicated in patients with known hypersensitivity to any of the components.

WARNINGS GARAMYCIN Opnthalmic Solution is not for injection, it should never be injected subconjunctivally, nor should it be directly introduced into the anterior chamber of the arm.

PRECAUTIONS Prolonged use of topical antibiotics may give rise to overgrowth of nonsusceptible organisms, such as fungi. Should this occur, or if irritation or hypersensitivity to any component of the drug develops, discontinue use of the preparation and institute appropriate therapy.

Ophthalmic ointments may retard corneal healing.

ADVERSE REACTIONS Transient irritation has been reported with the use of GARAMYCIN Ophthalmic Solution.

Occasional burning or stinging may occur with the use of GARAMYCIN Ophthalmic Solution.

DOSAGE AND ADMINISTRATION GARAMYCIN Ophthalmic Solution: instill one or two drops into the affected eye every four hours. In severe infections, dosage may be increased to as much as two drops once every hour.

GARAMYCIN Ophthalmic Ointment: apply a small amount to the affected eye two to three times a day.

HOW SUPPLIED GARAMYCIN Ophthalmic Solution—Sterile, 5-ml. plastic dropper bottle, sterile, boxes of one and six. Store away from heat.

GARAMYCIN Ophthalmic Ointment—Sterile, %-ounce tube, boxes of one and six. Store away from heat. PRECAUTIONS Prolonged use of topical antibiotics may

tube, boxes of one and six. Store away from heat

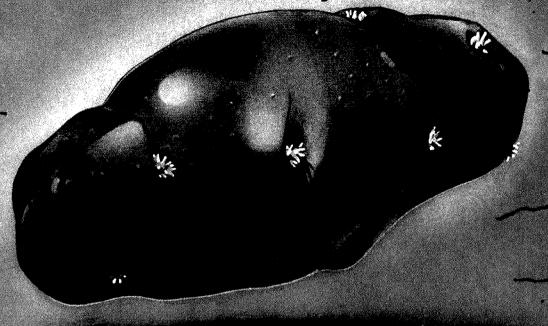
NOVEMBER 1973

For complete details, consult package insert or literature available from your Schering Representative; or Professional Services Department, Schering Corporation, Kenilworth, New Jersey 07033.

SWW-9110

Schering Corporation Kenilworth, N.J. 07033

Schering Ophthalmic Products aren't for all eyes...



But, when your patients have eye disorders ...

*conjunctivitis, corneal ulcer and other indications shown in product information

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suffonamide antibacterial for superficial ocular infections:

Sodium SULAMYD°

brand of SOCIUM SUITACETAMICE, USP OPHTHALMIC SOLUTION 30% STERILE OPHTHALMIC SOLUTION 10% STERILE OPHTHALMIC OINTMENT 10% STERILE



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1This drug has been evaluated as
possibly effective for this indication

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Specific products for specific needs

The Post Graduate Institute
of the
New York Eye and Ear Infirmary
announces
A PRACTICAL COURSE ON
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MONDAY, MARCH 20, 1978

DEFINITION CLASSIFICATION INSTRUMENTATION TECHNIQUE EVALUATION

A practical course on perimetry, with special emphasis on the proper use of the GOLDMANN type perimeter, for Ophthalmic Assistants.

Given under the direction of G. Peter Halberg, M.D., F.A.C.S. with the assistance of Phyllis Blumenthal, C.O., C.O.T.

Free: \$125.00 • Course limited to 8 students
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Ophthalmic Assistants, sponsored by an Ophthalmologist, are eligible.

For additional information, please write: Jane Stark, Registrar • Post-Graduate Institute New York Eye and Ear Infirmary 310 East Fourteenth Street New York, New York 10003

NATIONAL SPRING MEETING

at



APRIL 26-29, 1978

Ophthalmology Speakers:
Robert C. Drews, M.D., Clayton, Missouri
Jared M. Emery, M.D., Houston, Texas
Richard P. Kratz, M.D., Vn Nuys, Cal.
Bruce E. Spivey, M.D., San Francisco, Cal.
Bradley R. Straatsma, M.D., Los Angeles, Cal.

Otolaryngology Speakers:
William Jacquiss, M.D., Pittsburgh, Pa.
Donald Kamerer, M.D., Pittsburgh, Pa.
Walter Work, M.D., Ann Arbor, Mich.
Charles T. Yarington, M.D., Seattle, Wash.

Reservations: Write directly to The Greenbrier, White Sulphur Springs, West Virginia for hotel accomodations.

Advance Registration: Fee of \$225 required; checks payable to the West Virginia Academy of O & O. Send to: J. Elliott Blaydes, M.D., The Blaydes Clinic, Corner of Frederick & Woodland Avenue, Bluefield, West Virginia 24701.

AMA CREDIT CATEGORY I

HARVARD MEDICAL SCHOOL

Department of Continuing Education Announces a Course in GLAUCOMA

April 5, 6, and 7, 1978 at the

MASSACHUSETTS EYE AND EAR INFIRMARY

Under the Direction of

Paul A. Chandler, M.D., W. Morton Grant, M.D. and David K. Dueker, M.D.

This course, designed for ophthalmologists in clinical practice, will cover diagnosis and treatment of the various glaucomas. Emphasis will be placed on detailed case presentations, followed by open discussion by all participants on an informal basis.

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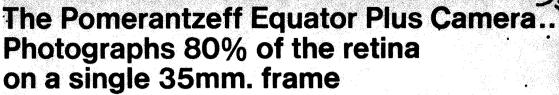
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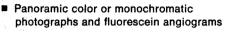
This course has Category I accreditation for 20 hours toward the AMA's Physicians' Recognition Award.

Fee: \$200 Registration: Please contact

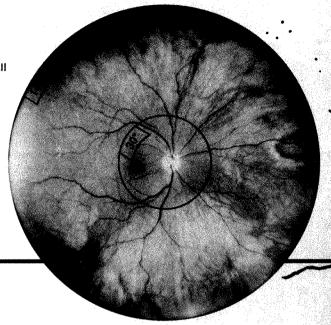
Department of Continuing Education, Harvard Medical School,

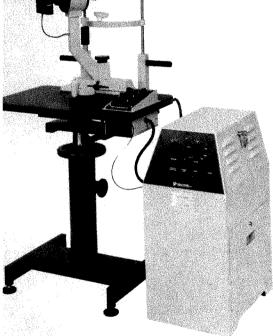
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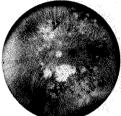




- Complete documentation of diabetic retinopathy, scleral buckles, tumors and all peripheral retinal diseases
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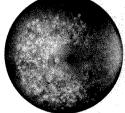


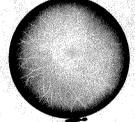




Diabetic Retinopathy

Malignant Melanoma





Pan-Retinal Photocoagulation

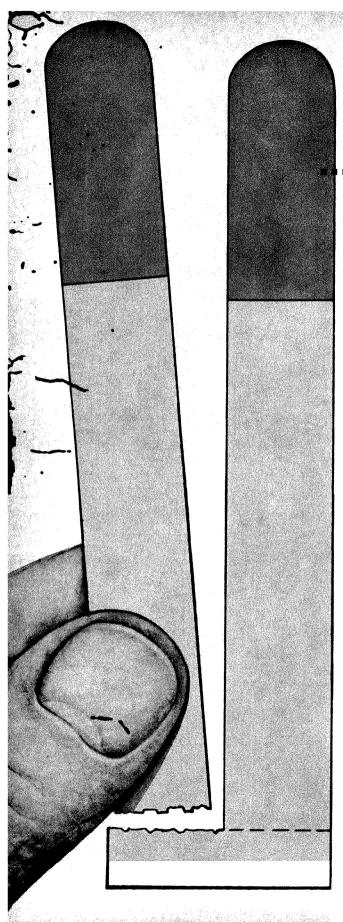
Fluoresgein Angiogram



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Protection against the risk of contamination, and possible infection, inherent in the use of sodium fluorescein solutions—one good reason for preferring sterile, individually wrapped, disposable FLUOR-I-STRIP-A.T.

Other good reasons: FLUOR-I-STRIP-A.T. is convenient and easy to use. With FLUOR-I-STRIP-A.T., you can control contact time, thus obtaining just the right amount of fluorescein needed. There's little chance of excess fluorescein to stain face or clothing, and the waterproof grip prevents staining your fingers.

Easy to use: simply retract the upper lid, apply the dry orange tip of the flexible applicator to the bulbar conjunctiva at the temporal side.



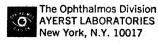


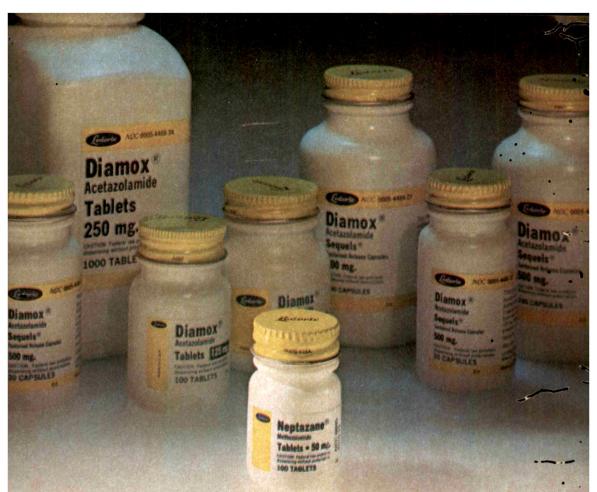
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Specially prepared applicators impregnated with a sodium fluorescein solution which also contains chlorobutanol (chloral derivative) 0.5%, polysorbate 80, boric acid, potassium chloride, and sodium carbonate.

Supplied: No. 1048—Boxes of 100 envelopes, each envelope containing two sterile strips.





In glaucoma, the last step before surgery when other agents fail

NEPTAZANE Methazolamide may be successful in glaucoma patients uncontrolled by acetazolamide in long-term therapy. In the long run of glaucoma, it may often turn failure into success.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: For adjunctive treatment of chronic simple (open angle) glaucoma, secondary glaucoma, and preoperatively in acute angle closure glaucoma where delay of surgery is desired in order to lower intraocular

pressure.

Contraindications: Severe or absolute glaucoma and chronic noncongestive angle closure glaucoma. Of doubtful use in glaucoma due to severe peripheral anterior synechiae or hemorrhagic glaucoma. Adrenocortical, hepatic, or renal insufficiency; electrolyte imbalance state, e.g., hyperchloremic acidosis; sodium and potassium depletion states.

Warnings: Although teratogenic effects demonstrated in rats at high doses have not been evidenced in humans, Methazolamide should not be used in

women of child-bearing potential or in pregnancy, especially in the first trimester, unless the expected benefits outweigh potential adverse effects.

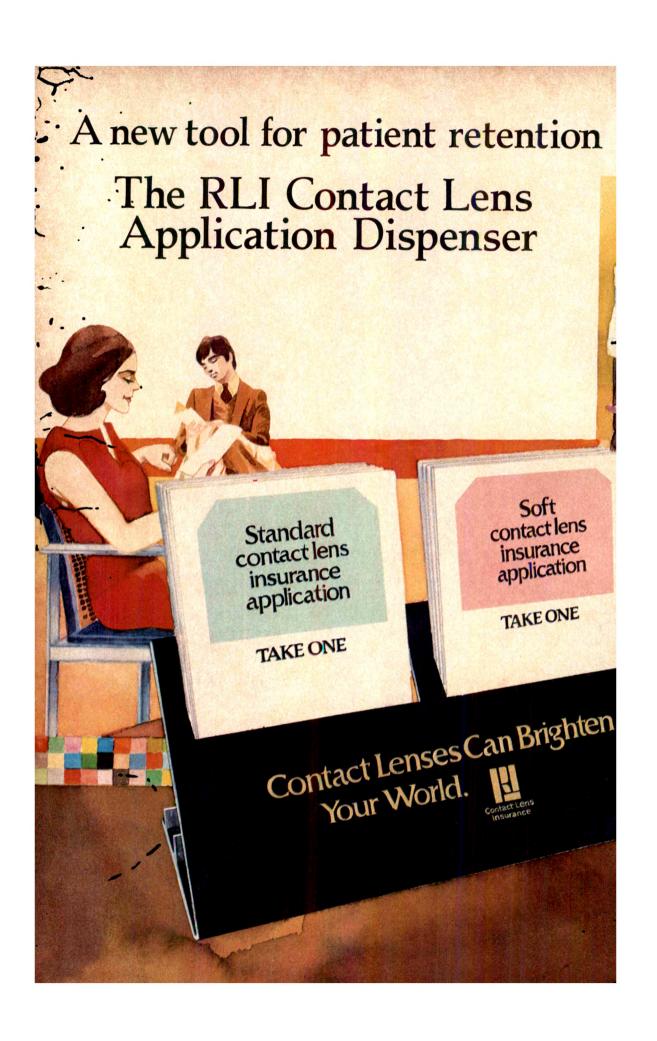
Precautions: Use with caution in patients with cirrhosis or hepatic insufficiency to forestall hepatic coma; those on steroid therapy; those with pulmonary obstruction or emphysema to avoid acidosis. Electrolyte balance should be maintained. Although not reported thus far with this drug, reactions common to sulfonamide derivatives, such as fever, leukopenia, hemolytic anemia, bone marrow depression or renal calculations.

Adverse Reactions (relatively mild and disappear or withdrawal or dosage adjustment): anorexia, nausea, vomiting, malaise, fatigue or drowsiness, headache; vertigo, mental confusion, depression, paresthesias. Urinary citrate excretion and uric acid output is decreased during use of this drug, but urinary calculi have not been reported.

Methazolamide TABLETS 50 mg. b.i.d. ort.i.d.



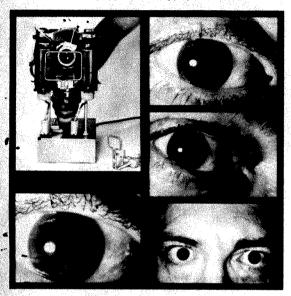
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Sodium SULAMYD®

brand of sodium sulfacetamide, U.S.P.
Ophthalmic Solution 30%. Solution 10%, Ointment 10% — Sterile
INDICATIONS Sodium SULAMYD is indicated for the treatment
of conjunctivitis, corneal ulcer, and other superficial ocular infections due to susceptible microorganisms, and as adjunctive treatment in systemic sulfonamide therapy of trachoma.
CONTRAINDICATIONS Hypersensitivity to sulfonamide preparations contraindicates the use of Sodium SULAMYD.
PRECAUTIONS The solutions are incompatible with silver
preparations. Ophthalmic ointments may retard corneal healing
Non-susceptible organisms, including fungi, may proliferate with
the use of these preparations. Sulfonamides are inactivated by the
para-aminobenzoic acid present in purulent exudates. Sulfonamide
sensitivity reactions may occur.

June 1972 9838358

METIMYD®

brand of prednisolone acetate, U.S.P., and sulfacetamide sodium, U.S.P.

Ophthalmic Suspension and Ointment—Sterile Each ml. or Gm. contains 5 mg prednisolone acetate and 100 mg sulfacetamide

INDICATIONS — Suspension Based on a review of this drug by the National Academy of Sciences — National Research Council and/or other information. FDA has classified the indications as follows:

"Possibly" effective for the treatment of the following inflammatory and allergic conditions affecting the eyelids and anterior segment of the eye. EYELIDS, allergic blepharitis, blepharitis associated with seborheig dermatitis and other incomprutent forms of conjunctivitis including those associated with hay fever and conjunctivitis due to physical agents such as foreign bodies, chemicals (acids, alkalies) and other inflatis. CORNEA SCLERA, IRIS, AND UVEA interstitial, postoperative, and sclerosing keratitis; chemical and thermal burns of the cornea; corneal ulcer, herpes zoster ophthalmicus; phylictenular keratoconjunctivitis; corneal neovascularization, scleritis; episcleritis; acute, chronic, and traumatic indocyclitis. Final classification of the less-than-effective indications requires further investigation.

Ointment METIMYD is indicated in the following inflammatory and allergic conditions affecting the eyelids and anterior segment of the eye:

Eyelids allergic blenharitis blenharitis

of the eye:

Eyelids allergic blepharitis: blepharitis associated with seborrheic dermatitis, other nonpurulent types of blepharitis.

Conjunctiva allergic conjunctivitis, ie, vernal, phylotenular, and other nonpurulent forms of conjunctivitis including those associated with hay fever, conjunctivitis due to physical agents such as foreign bodies, chemicals lacids, alkalies) and other irritants.

Cornea, Sclera, Iris, Uvea interstitial, postoperative, and sclerosing keratitis, chemical and thermal burns of the cornea; corneal ucer, herpes zoster ophthalmicus; phylotenular keratoconjunctivitis, corneal neovascularization; scleritis; episcleritis; acute, chronic, and traumatic indocvclitis.

corneal neovascularization, scleritis, episcleritis; acute, chronic and traumatic indocyclitis. In deep-seated infections, such as endophthalmitis, panophthalmitis, and orbital cellulitis, or when systemic infection threatens, specific oral (antibiotic, sulfonamide) therapy should be employed local treatment may be used as adjunctive therapy. CONTRAINDICATIONS The contraindications for METHMYD are the same as those for other corticosteroid-sulfonamide preparations. Topical ophthalmic corticosteroid preparations are contraindicated in, early acute herpes simplex and the early acute stages of most other viral diseases of the cornea and conjuctiva; active tuberculosis of the anterior segment of the eye fungal disease of the cornea, conjunctiva and lids; acute purulent untreated infections of the eye which, like other diseases caused by microorganisms, may be masked or enhanced by the presence of the steroid, individuals with known sensitivity to any of the ingredients.

of the steroid; individuals with known sensitivity to any of the ingredients.

PRECAUTIONS Extended use of topical steroid therapy may cause increased intraocular pressure in certain individuals. In prolonged therapy, it is advisable that intraocular pressure be checked frequently. In those diseases causing thinning of the cornea, performance has been known to have occurred with the use of topical steroids. As with any antibacterial preparation, prolonged use may result in overgrowth of non-susceptible organisms, including fungit superinfection occurs, appropriate measures should be instituted. Sensitivity develop, discontinue use.

The protracted use of topical corticosteroids in the eye reportedly has been rarely associated with the development of posterior subcapsular cataracts.

January 1973, July 1973 9835356, 10106524

January 1973, July 1973, 9835356, 10106524



Schering Corporation Kenilworth, N.J. 07033

SWW-9570



CHOICE FOR INITIALTH

r efficacy – against conjunctivitis and other in-tions of external eye and adnexa due to a wide age of susceptible pathogens.

nge of susceptible pathogens.

Gram-negative: susceptible strains of: H. influene; E. coli; K. pneumoniae; M. lacunata; Enterobacaerogenes (formerly Aerobacter); H. aegyptius
d Neisseria sp., including N. gonorrhoeae.

Gram-positive: susceptible strains of: staphylocci and streptococci, including D. pneumoniae

Problem pathogens: susceptible strains of: P. aeru-Problem pathogens: susceptible strains of: *P. aeru-ginosa* and *Proteus* sp. (indole-positive and -negative).

for gentle potency – generally avoids sensitivity reactions and irritation.

for reliability – no significant organism resistance to date. This may occur in the future.

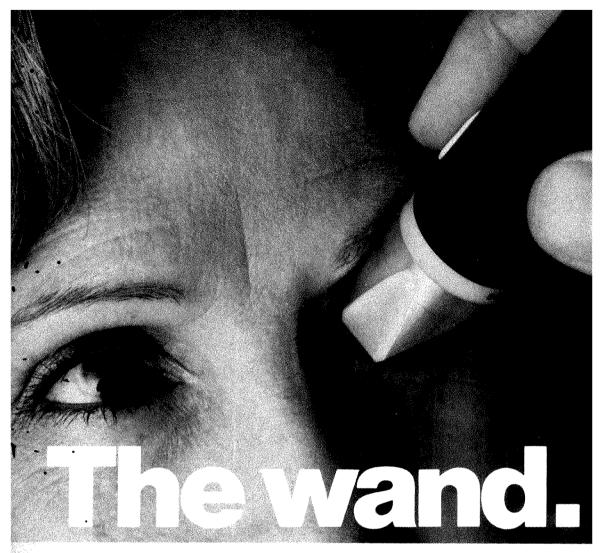
road spectrum activity against many ram-negative and gram-positive organisms

entamicin sulfate, U.S.P.

th ml. or gram contains gentamicin sulfate equivalent .0 mg. gentamicin.

to susceptible pathogens se see product information on facing page Solution-Sterile Ointment-Sterile

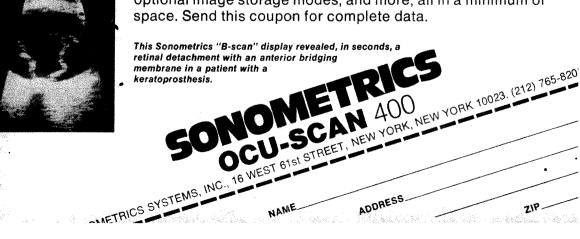
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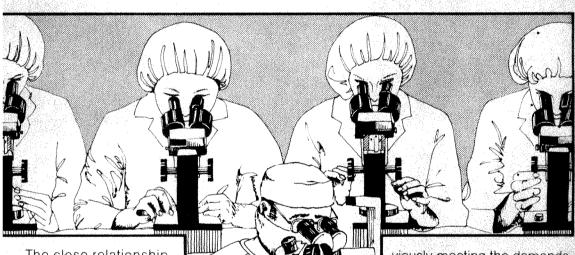
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viously meeting the demands of the implant surgeon, but due to technological advancement, has been found not to measure up to those presently manufactured by us

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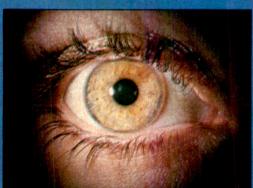
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Aquaflex® (tetrafilcon A) Hydrophilic Contact Lens





Outstanding Visual Acuity



Optimum Centration



Predictable Fit

h Performanc Stor

Soft contact lens performance that meets your exacting professional needs.

Dutstanding Visual Acuity

\ major benefit of Aquaflex® (tetrafilcon A) tydrophilic Contact Lenses is excellent visual icuity. Clinical data revealed that 88% of the vearers achieved 20/20 vision or better, with 15% attaining 20/25 or better. Practitioners eported that Aquaflex lenses often corrected nore astigmatism and gave sharper vision har other soft lenses. This quality of performance is due to a unique combination of sophisticated lens design, special lens naterial and the "Vault" system of fitting.

Optimum Centration

A clinical study showed that over 96% of Aquaflex contact lens wearers achieved optimum centration. By centering so well, Aquaflex lenses virtually eliminate the induced astigmatism and lessened visual acuity esulting from decentered lenses.2 Precise centration helps in attaining a good fit with full corneal coverage and maximum visual acuity, while permitting proper corneal respiration.

Predictable Fit

No complicated graphs, charts and calculations, because the Aquaflex "Vault" system reduces fitting variables to just one... the lens "Vault". Only 5 Vaults are needed to fit Aquaflex lenses;3 and in clinical studies more han 90% of the patients were fitted with just 3 of these Vaults. The bicurve, lathe-cut lens construction, with spherical front and back surfaces, promotes reliable fit independent of power; and the large posterior optical zone gives stability of vision with lens movement.

Additional Advantages

Other significant attributes of the Aquaflex® (tetrafilcon A) lens, contributing to practitioner success and patient acceptance, are: reliable over-refraction,^{2,4} ease in handling, durability, excellent reproducibility² and exceptionally high standards of quality assurance, including 100% wet inspection. A study has shown that less than 5% of lenses shipped were returned because of questionable performance, discomfort, damage or defects. The fitting procedure is simple and fast. You can fit from a small diagnostic set, or use a dispensing inventory. Lenses may be ordered by a toll-free phone call, and UCO Optics' service is quick and dependable. More and more practitioners are using Aquaflex lenses every day. Shouldn't you?

Hydrophilic Contact Lens

For more information and a copy of the Professional Fitting Guide, mail the coupon. Or, phone toll-free 800-828-4580 (in N.Y.State 800-462-4332).

There sed in Adulated Representation of the Interest of the In Library North Representative call

- 1. Data on file: UCO Optics, Inc., Scottsville, N.Y. 14546 2. Morrison, Robert J.: International Contact Lens Clinic,
- 3. Gruber, Ellis & Gordon, Stanley: Contact Lens Forum, February 1977
- 4. Greenspoon, Morton K.: Contact Lens Forum, November 1977.

*Aquatlex is a registered trademark of UCO Optics, Inc.



DESCRIPTION

DESCRIPTION

AQUIAFLEX* (letraflicon A) Hydrophilic Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The lens material, letraflicon A, is a hydrophilic random terpolymer of 2-hydroxyethylmethacrytalis. N-vingft-2-pyriolidone and methylmethacrytalis. The polymer is a three-dimensional network of terpolymer chains joined by divinythenzene crosslinks. It consists of 55.5% letraflicon A and 42.5% water by weight when they hydrated in normal saline solution. Lenses have a nominal diameter of 13 mm.

ACTIONS
In its hydrated stafe the AQUAFLEX* Hydrophilic Contact Lens is soft and pliable. When dry, the lens becomes hard and brittle. These states are completely reversible and a lens which has been permitted to dry out will recover all of its hydrated properties when placed in normal saline for a period of two hours. When placed on the human cornea the hydrated lens acts as a refracting medium to compensate spherical ametropias. The material has a refactive index of 1.43 and the lens has a visible light transmittance greater than 97%.

1

ACMAFLEX* Hydrophilic Contact Lenses are indicated for the correction of vision in persons with non-diseased eyes who have spherical ametropias, corneal astig-matism of 2.50 clopters or less and/or refractive astigmatism of 2.00 cliopters

ACULAFILEX* Hydrophilic Contact Lenses are contraindicated in the presence of any of the following conditions: (1) Acute and subacute inflammation of the anterior segment of the eye. (2) Any eye disease which affects the cornea or conjunctiva. (3) Insufficiency of lacingais secretion. (4) Corneal hyposethesia. (5) Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS

Medications and Eye Drops: AQUAFLEX* Lenses must be stored in an appropriate solution when off the eyes, the type of solution being dependent on the system used for disinfection. When the lenses are disinfected with the thermal disinfection system, they may be stored only in BOILInSOAK* Solution (sterile buffered isotonic solution containing boric acid, sodium borate, sodium chloride 0.7%, preserved with Thimerosal (Lilliy) 0.01% and edetate disodium 0.1%). When it lenses are disinfection with the chemical disinfection system, they may be stored only in FLEXSOL* Disinfection and Storage Solution (sterile buffered isotonic solution of sodium chloride, sodium borate, boric acid, polyvinjetyprolidone, polyoxyethylene and polyoxypropylene, preserved with Thimerosal (Lilly) 0.001%, chlorhexidine 0.05% and edetated disodium 0.1%).

No ophthalmic solutions or medications, including hard contact lens solutions, can be used by AQUAFLEX* Lense wearest prior to or while the lenses are in place on the eyes. Also, no solutions, including hard contact lens solutions, other than BOILnSOAK* Solution or FLEXSOL* Solution may be used on AQUAFLEX* Lenses, when they are off or the eyes.

Since liquids and vapors may be absorbed by AQUAFLEX* Lenses, they should not be placed in the mouth for wetting, nor wiped with a cloth or issue.

Abrasions and infections: If a lens becomes less confortable than it was when list placed on the wearer's comea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present, or any abnor malley econdition is observed concurrently with lens wear, the lens should be removed immediately and a physician consulted.

Wearing Restrictions: AQUAFLEX* (tetraflicon A) Hydrophilic Contact Lenses should not be worn while swimming, sleeping, or in the presence of irritating tumes

Visual Blurring: If visual blurring occurs, the lens must be removed until the con-

Lens Care Regimen: Patients must adhere to the recommended daily care procedures for AQUAFLEX* Hydrophilic Contact Lenses. Failure to follow this procedure may result in the development of serious ocular infections.

PRECAUTIONS

Storage: ACUAFLEX* Lenses may be stored only in the appropriate storage solution: BOILnSOAK* Solution or FLEXSOL* Solution depending on disinfecting methods used. If left exposed to air, the ienses will dehydrate, become brittle and break readily. If a lens dehydrates, it should be soaked in either BOILnSOAK*
Solution or FLEXSOL* Solution until it returns to a soft, supple state.
Cleaning and Disinfecting: ACUAFLEX* Lenses must be both cleaned and disinfected daily. Separate procedures and products are needed to clean and to disinfect. Two methods of disinfection, thermal or chemical, have been shown to be equally effective. The choice of disinfection system should be made in consultation with your eye care practitioner.

Cleaning: Daily cleaning is necessary to remove mucus and other deposits which may have accumulated on the lens surface. Each time the lenses are removed from the wearer's eyes, both surfaces of the lenses must be cleaned using several drops of PREFEX* Cleaning is necessary to remove after isotrotic aqueous solution consisting of sodium phosphates, sodium chloride, tyloxapol, hydroxyethyl-cellulose and polynnyl atorib with Thimscroal (Lilly) to 0.04% and ecleate disordium 0.2% added as preservatives). Lenses must be cleaned before they are disinfected, as deposits on the lenses tend to harden and become more difficult to remove after the lenses are disinfected.

Disinfecting: AQUAFLEX* Lenses may be disinfected with either a heat or chemical regimen. One method or the other must be selected, but not both. The user must not afternate between methods.

must not alternate between methods.

Triust not alternate perween methods. Thermal Disinfection Method: AQUAFLEX* Lenses may be effectively disinfected after cleaning with PREFLEX* Cleaning Solution with use of the AQUASEPT* Patient Unit and BOILINSOAK* Solution. Fresh BOILINSOAK* Solution must be used for daily storage of lenses or each time the lens is stored. The AQUASEPT* Patient Unit requires distilled water. The AQUAFLEX* Lens Storage Containers must be empired and filled with fresh BOILINSOAK* Solution just prior to disinfecting the lenses.

Chemical Disinfection: Disinfection with PREFLEX* Cleaning Solution, FLEXSOL* Solution and NORMOL* Rinsing Solution (sterile buffered isotophic aqueous solution of sodium chloride, sodium borate and boric acid, preserved with Thirmerosal (Lilly) 0.001%, dedated isosolum 0.1% and chlorhexidine 0.005%) has also been shown to be an effective disinfection system for daily care of AQUAFLEX* Lenses. AQUAFLEX* Lenses must be cleaned and rinsed daily for after wearing) with PREFLEX* Cleaning Solution and NORMOL* Rinsing Solution. The AQUAFLEX* Lense Storage Containers must be emptied and refilled with fresh. FLEXSOL* Solution each time the lens is stored. Fresh FLEXSOL* Solution must be used daily for storage and disinfection. WARNING: DO NOT MIX OR ALTERNATE THE DISINFECTION AND STORAGE SYSTEMS. FLEXSOL* SOLUTION SHOULD NOT BE USED WITH HEAT.

Anough Not Be USED WITH HEAT.

Hyglene: Before handling the lenses, hands must be washed, rinsed thoroughly and dried with a lint-free towel. Cosmetics, lotions, soaps, oils and hand creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

are yeary reas senteru. Fluorescelin: Never use fluorescein while the patient is wearing the tenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with normal saline solution and wait at least one hour before replacing the lanses. Too early replacement may allow the lenses to absorb residual fluorescein.

ADVERSE REACTIONS

Serious corneal damage may result from wearing lenses which may have soaked in hard contact lens solutions. Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will refleve the irritation.

Very rarely a lens may adhere to an eye as a result of a pattent sleeping with the lens on, or as a result of wearing a hypotonic lens. If a tens adheres for any reason, the patient may be instructed to apply a few drops of BOLINSOAK® Solution (if using a thermal disinfection regimen) or ADAPETTES* Lubricating Solution (furthered isotonic aqueous solution containing ADSORBOBASE® (polyvinyl-pyrrolidone with other water soluble polymers) with Thimerosal (Lilly) 0.004% and edetate disodium 0.1% added as preservatives) (if using a chemical disinfection regimen), and wait until the lens moves freely before removing it.

Clinical studies indicate that corneal edema as manifested by symptoms such as rainbows or halos around light or visual blurring may occur if lenses are worn continuously for too long a time. Removal of the lenses and a rest period of at least one hour generally relieve these symptoms. If symptoms do not subside promptly, professional consultation should be obtained.

Excessive tearing, unusual eye secretions and photophobia are not normal; if these symptoms occur, the patient should be examined to determine their cause.

DOSAGE AND ADMINISTRATION

DOSAGE AND ADMINISTRATION

Fitting: Conventional methods of fitting contact lenses do not apply to AQUAFLEX* (tetraflicon A) Hydrophilic Contact Lenses. For a detailed description of the fitting technique, refer to the Professional Fitting Guide for AQUAFLEX* Hydrophilic Contact Lenses, copies of which are available from: UCO Optics, Inc., Scottsville, New York 14546.

Wearing Schedule: There may be a tendency for the patient to overwear the lenses initially. Therefore, the importance of adhering to the following initial daily wearing schedule should be stressed to the patient.

Day	Wear Time (Hours)	Rest Period Wear Time (Hours) (Hours)		vvear i me Hest renog	
1	4	2	4		
2	4	2	4		
3	5	2	5		
4	6	2	5		
5	7	2	5		
6	7	1	6		
7	8	1	7		
8	8	1 2	8		
9	9	1 .	8		
10-14	10	4	balance of		
15	all waking hours		waking hourst		

tienses should never be worn 24 hours a day

Catalog No. A0101

Lens Care and Handling: Care must be taken on the initial visit to assure that the patient is supplied with an appropriate AQUAFLEX* Patient Care Kit and fully understands all care and handling instructions for the lenses. As with any contact lens, regular recall visits are necessary to assure patient health and compliance with instructions.

How Supplied: Each lens is supplied sterile in a glass vial containing normal saline solution. The glass vial is marked with the vault number, dioptric power, and manufacturing identification number.

The AQUAFLEX® Patient Care Kit is required for lens cleaning, disinfection and storing of the lenses. The Kit may consist of either of the following: Thermal Disinfection Regimen

AQUAFLEX* Patient Care Kit AQUASEPT* Patient Unit

AQUAFLEX* Lens Storage Container		Catalog No. A0201
PREFLEX® Cleaning Solution		Catalog No. A1201
BOILnSOAK* Solution		Catalog No. A1301
AQUAFLEX* Patient Instruction Book		Catalog No. X0102
Chemical Disinfection Regimen		33
AQUAFLEX* Patient Care Kit		September 1987
AQUAFLEX* Lens Storage Container		Catalog No. A0202
PREFLEX* Cleaning Solution		Catalog No. A1201
NORMOL* Rinsing Solution		Catalog No. A1401
FLEXSOL* Disinfection and Storage Solution		Catalog No. A1501
ADAPETTES* Lubricating Solution		Catalog No. A1601
AQUAFLEX* Patient Instruction Book		Catalog No. X0102

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CAUTION: Federal law prohibits dispensing without prescription.

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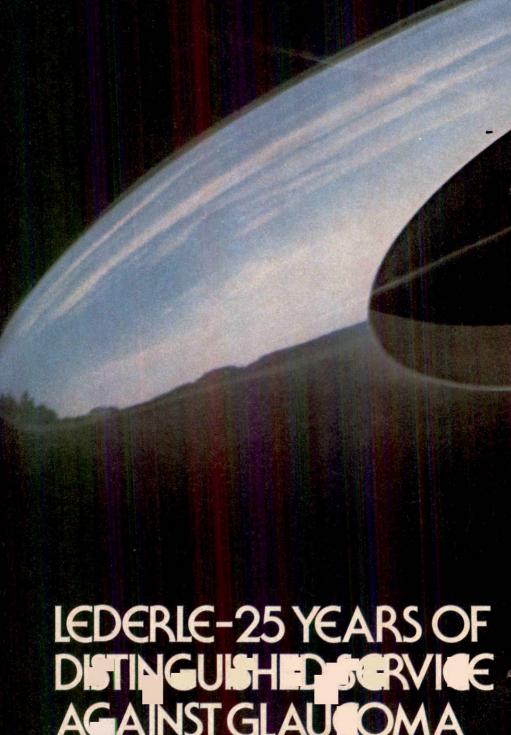
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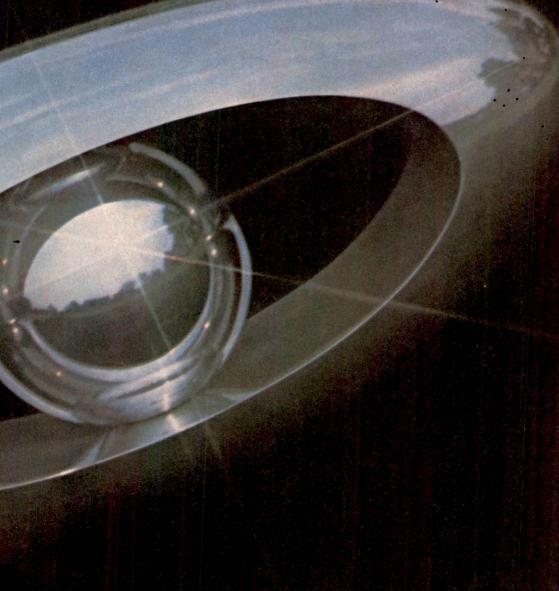
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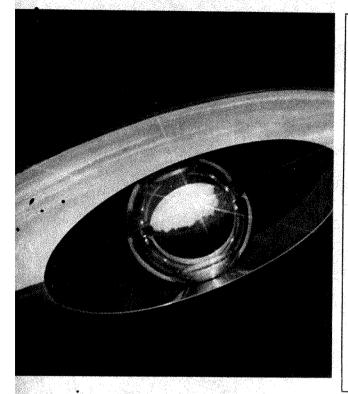
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GLAUCOMA

MARCH 3-4, 1978 SALT LAKE HILTON HOTEL SALT LAKE CITY, UTAH

FACULTY

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This meeting will concentrate on the practical aspects of the glaucomas. Diagnosis will be covered but management will be stressed. Two panel discussion periods are scheduled. Physicians may obtain 8 hours of Category I Continuing Medical Education credit for attending this course.

medical Education credit for attending this course. Registration fee is \$75.00 for practitioners, and \$50.00 for residents upon application from their Department Head. Mail registration fee, payable to "University of Utah" to Mrs. Jean Florence, Division of Ophthalmology, University Medical Center, Salt Lake City, Utah, 84132. Make hotel reservations directly with Salt Lake Hilton Hotel. 150 W. 500 So. Salt Lake City, Utah, 84101. Early registration and hotel reservations are advised, March is the height of ski season in Utah.

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Acetazolamide

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All forms: Chronic simple (open angle) glaucoma, secondary glaucoma, and preoperatively in acute angle closure glaucoma where delay of surgery is desired in order to lower intraocular pressure.

Contraindications: When sodium and/or potassium serum levels are depressed, in marked kidney and liver disease or dysfunction, suprarenal gland failure and hyperchloremic acidosis. Long-term use in chronic noncongestive angle closure glaucoma.

Warning: Although teratogenic and embryocidal effects demonstrated in mice at more than ten times the equivalent therapeutic doses have not been evidenced in humans, do not use DIAMOX in pregnancy, especially during the first trimester, unless expected benefits outweigh these potential adverse effects.

Precautions: Increasing the dose may increase drowsiness and paresthesia and decrease diuresis. Adverse reactions common to all sulfonamide derivatives may occur: fever, rash, crystalluria, renal calculus, bone marrow depression, thrombocytopenic purpura, nemolytic anemia, leukopenia, pancytopenia, agranulocytosis. Early detection is advised and if such occur, discontinue drug and institute appropriate therapy.

Adverse Reactions: Short-term therapy: (minimal) paresthesias, particularly a "tingling" feeling in the extremities; some loss of appetite, polyuria, drowsiness, confusion. Longterm therapy: An acidctic state may superviewe usually corrected by bicarbonate. Transient myopia.

Other: (occasional) urticaria, melena, hematuria, glycosuria, hepatic insufficiency, flaccid paralysis, convulsions.

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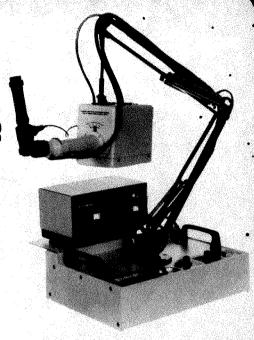
Willi Ott Kontaklinsen Stauffacherstrasse 5, 8004 Zurich, Switzerland

DMV Contact Lens Company
Box 2829 Zanesville, Ohio 43701

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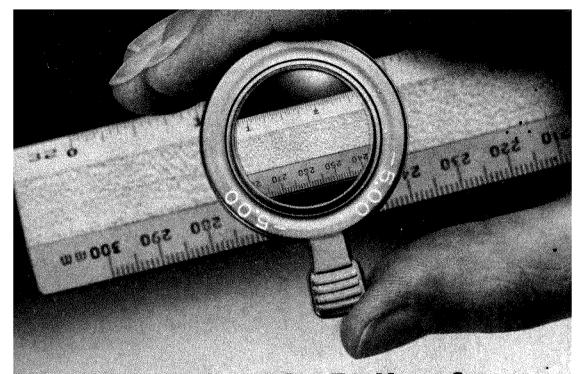
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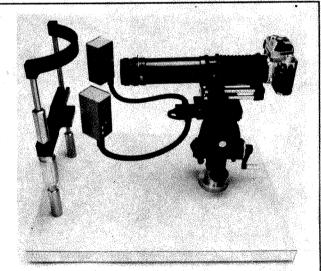
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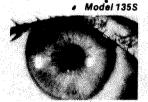
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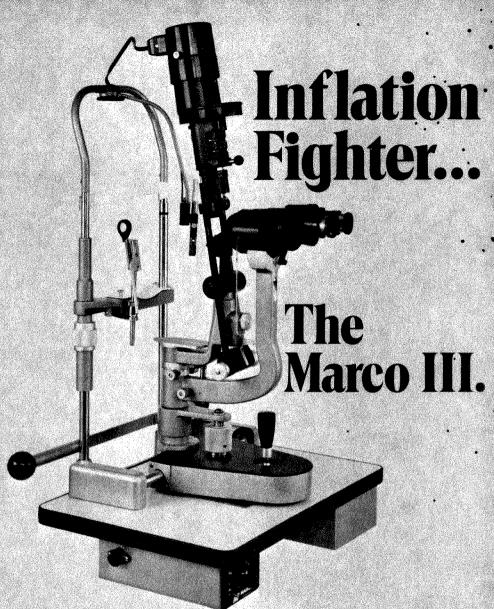
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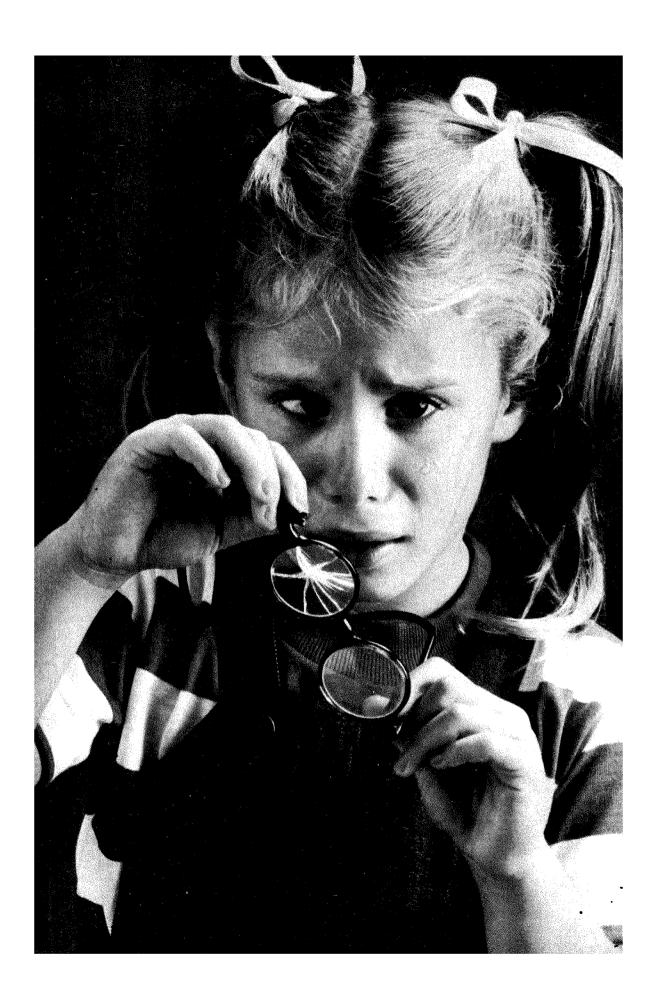
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6. Anticholinesterase fit ins should be used with avtragree.

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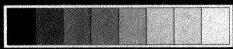
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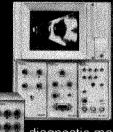
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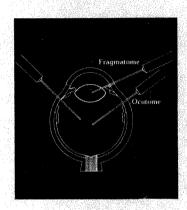


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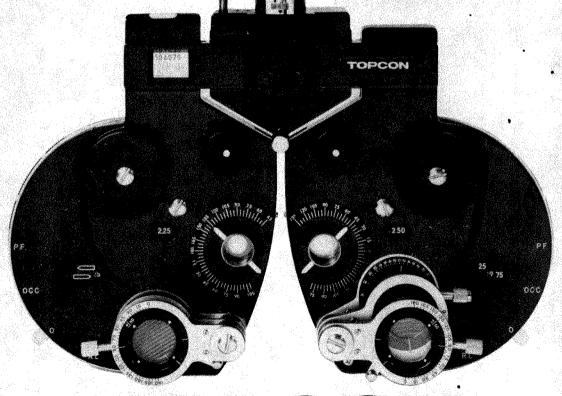
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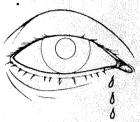


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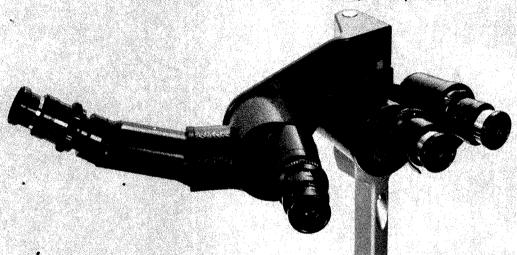
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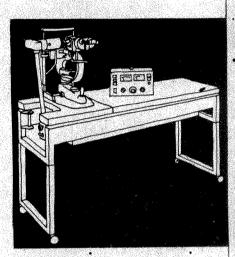
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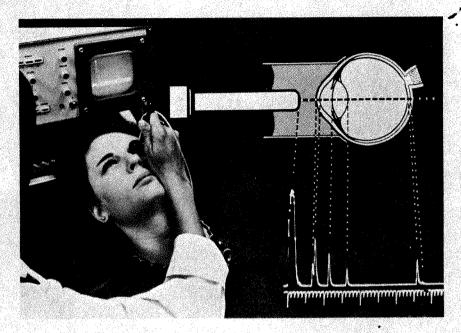
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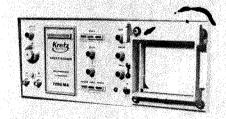
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(To be presented in conjunction with the 4th Annual DermatoCryosurgical Seminar and to be proceeded [March 9-11, 1978] by the 18th Annual Instructional Course in Contact Lens Fitting by the Ophthalmologist and followed by the 1st Annual American College of Cryosurgery Program, March 13, 1978)

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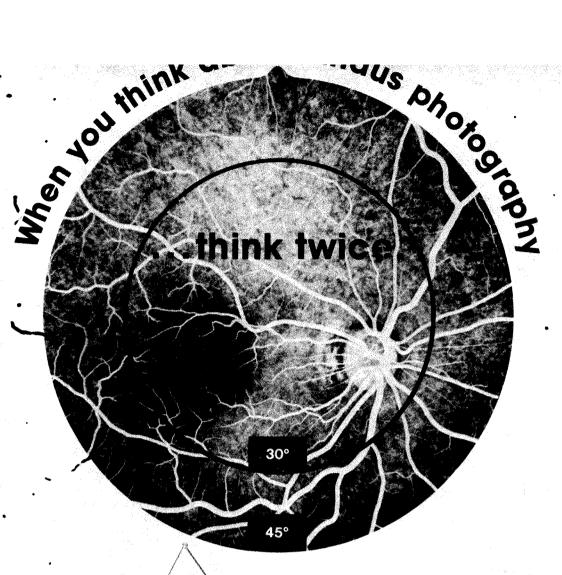
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> REFERENCE NOTES: 1. Leibowitz, H. M. and Kupferman, A. Bioavailability and therapeutic effectiveness of topically administered corticosteroids. Trans Am Acad Ophthalinol Otolaryngol, 79 (1): op 78-op 88, 1975. 2. Ibid. 3. Ibid.

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OPTIC ATROPHY AND GLAUCOMATOUS CUPPING

RONALD L. RADIUS, M.D., AND A. EDWARD MAUMENEE, M.D.

The cause of glaucomatous disk and field changes remains enigmatic. Many theories have been advanced to explain progressive optic atrophy, disk cupping, and visual field loss characteristic of this disease. One currently popular hypothesis suggests that mechanical compression of neuronal axons, possibly at the lamina cribrosa, results in axonal death and optic atrophy. ¹⁻⁶ Progressive nerve head cupping is considered a direct consequence of increased intraocular pressure (IOP).

An alternative suggestion holds that increase in IOP compromises ocular perfusion pressures at the optic nerve head, precipitating local vascular collapse, chronic neuronal ischemia, and ultimately optic atrophy.^{7–13} Progressive disk cupping with axonal and glial tissue loss occurs in response to prolonged ischemia.

Various experimental and clinical observations have been made in support of both of these hypotheses; however, exact mechanisms involved in progressive cupping and field loss remain unsettled. 4-6,10,12-16 We compared optic atrophy of various causes with optic atrophy in glaucoma. We also reviewed nerve head pallor, cupping, and visual field function.

All patients were drawn either from our personal case histories or from chincal records here. All cases reviewed had been coded as either unilateral or bilateral optic atrophy. Glaucoma patients with characteristic disk and field changes were excluded from study. We also excluded patients with "low tension" glaucoma who had normal IOP, characteristic disk and field pathology, and no other demonstrable cause for their optic atrophy. We included only patients who could be examined by us or whose records were complete, including stereoscopic or color photographs of the optic nerve heads.

In patients examined, we evaluated clinical history, visual function, field defects, and physical examination to best establish the likely diagnosis of their optic atrophy. Slit-lamp biomicroscopy was used to examine optic nerve heads. Disk pallor and clinical examinations were evaluated to substantiate the diagnosis of optic atrophy. Extent of optic nerve head cupping was quantified by measuring vertical and horizontal meridians of the physical cup:disk ratio and recording average values. Where color photographs were available, we compared these measurements with those obtained after study of projected images of optic nerve heads. Analysis of stereoscopic color photographs was utilized to determine the extent of physical cupping of the nerve head. Cases of patients unavail-

MATERIAL AND METHODS

From the Wilmer Ophthalmologic Institute, Johns Hopkins Hospital, Baltimore, Maryland.

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able for physical examination were reviewed by using medical chart data and color photographs of their optic nerve heads.

All data demonstrating extent of nerve head cupping are presented as mean cup: hisk ratio ± one standard deviation of the mean. Statistical analyses reflect significance of differences between mean optic nerve head cupping in eves with optic atrophy compared to that in contralateral uninvolved eyes. We performed a second analysis by combining all patients with either unilateral or bilateral disease. Mean cup: disk ratio of this population was compared to that of eyes of 50 diabetic patients previously screened for proliferative retinopathy. Fundus pathology in this group was limited to mild background retinopathy or no apparent disease.

In all applyses, Student's t-test for statistical significance was used. In instances where direct comparison with contralateral eyes was possible, we performed paired analyses.

RESULTS

We reviewed a total of 112 cases. Optic atrophy resulted from various causes, including ischemia, trauma, demyelinating disease, pressure necrosis, and congenital lesions. There were 55 women and 57 men. Ages ranged from 5 to 88 years; mean age was 45 ± 19 years. As judged by clinical history and previous examinations, duration of pathology ranged from one year to 54 years. Mean duration of symptomatology was 8 ± 13 years. We studied a total of 170 eyes with optic atrophy; 30 cases were in the right eye, 24 in the left, and 58 were bilateral.

Mean cup:disk ratio of all eyes examined was 0.35 ± 0.15 ; range was from 0.10 to 1.00. Mean-cup:disk ratio of eyes of 50 diabetic patients with minimal fundus pathology was 0.30 ± 0.12 , with a range from 0.10 to 0.80. Physical cupping of the

optic nerve head as determined in this study was significantly greater in eyes with optic atrophy than that seen in our control sample (P< .05).

When cases of patients with unilateral disease were analyzed separately, mean physical cupping was 0.35 ± 0.13 in eyes with nerve atrophy and 0.31 ± 0.14 in contralateral normal eyes. Eyes with optic atrophy had a small but significant increase in physical cupping of the optic nerve head (P < .05).

We classified each of 112 patients into one of six possible groupings reflecting various causes of optic atrophy. These categories included ischemic, traumatic, demyelinating disease, pressure necrotic, congenital, and miscellaneous causes of optic atrophy. Statistical analyses were repeated for each of these separate groups of patients.

Ischemia—Ischemia was the cause in 21 cases of optic atrophy. Five patients studied had bilateral disease, so 26 eyes were reviewed. Eleven eyes had ischemic optic neuropathy with sudden loss of vision, altitudinal field defects, and systemic hypertension. Five eyes developed optic atrophy, presumably on an ischemic basis, after uneventful cataract surgery. Four patients had had attacks of giant cell arteritis with ocular involvement and six patients had suffered occlusion of their central retinal artery.

Ages ranged from 14 to 74 years of age with a mean of 54 ± 17 years. Duration of optic atrophy was from one to 20 years with a mean of 3 ± 5 years. Mean cup: disk ratio of affected eyes was 0.28 ± 0.18 . This value was not significantly different from that recorded in eyes of 50 diabetic patients (0.30 ± 0.12) . When paired analyses were performed comparing atrophic nerve heads with those of normal contralateral eyes, differences were again not statistically significant (Table).

In most cases, diseased eyes demonstrating significant pallor of the optic

TABLE
Cup:disk ratio in patients with optic atrophy

	No. of	No. of	Age	Duration		Cup Disk Ratio	
Cause	Cases	Eyes	(yrs)	(yrs)	Affected Eyes	Unaffected eyes	Control Eyes
Ischemia	21	26	54±17	3.0±5.0	0.28±0.18	0.30±0.12	0.30±0.12
Trauma	$\overline{13}$	16	38±15	4.3±5.0	0.32±0.13	0.28±0.11*	0.30 ± 0.12
Inflammation	23	35	44±18	14±14	0.30_0.11	0.30 ± 0.10	0.30±0.12
Compression	8	12	55 ±7	3.2±4.1	0.29±0.09		0.30_0.12
Congenital	27	41	25±18	25±18	0.35 ± 0.12	<u> </u>	0.30±0.12*
Miscellaneous	20	30	56±21	10±8	0.37 ± 0.22	0.30±0.08*	0.30±0.12
All cases	112	170	45±19	8±13	0.35±0.15	0.31±14*	0.30±0.12*

^{*}P<.05. Statistical significance reflects analysis of differences between mean cup:disk ratio of eyes with optic atrophy compared to that of normal contralateral eyes or eyes of diabetic patients with minimal for the pathology.

nerve head showed little if any increase in cupping. A typical case was that of a 65-year-old white woman who had had a central retinal artery occlusion involving the left eye. Although there was a slightly larger cup in the left eye compared to that of the right eye (Figs. 1 and 2), there was no further progression of asymmetry four years after vascular occlusion. Further-

more, disk changes did not appear to be glaucomatous in nature.

Of 26 eyes reviewed, only one demonstrated optic nerve head cupping at all resembling that seen in glaucoma. That was in the case of a 48-year old black woman noted to have decreased visual acuity, altitudinal field loss, and systemic hypertension consistent with a diagnosis of ischemic optic neuropathy. Examination of her right eye revealed marked op-

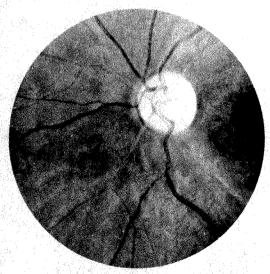


Fig. 1 (Radius and Maumenee). Central retinal artery occlusion in a patient's left eye. Note a slight increase in cup; disk ratio in the affected eye compared to that of the normal contralateral eye in Figure 2.

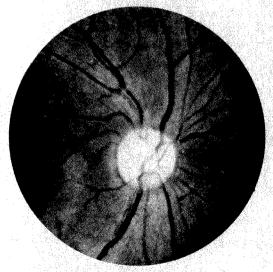


Fig. 2 (Radius and Maumenee). Fundus photograph of the normal contralateral right eye of the patient in Figure 1.

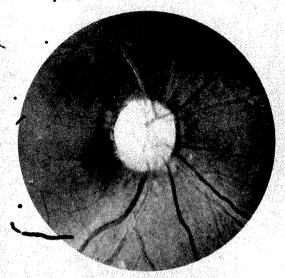


Fig. 3 (Radius and Maumenee). Fundus photograph of the right eye of a 45-year-old patient with ischemic optic neuropathy. Note marked pallor of the optic nerve head and enlarged cup:disk ratio of 0.9.

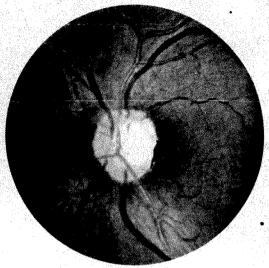


Fig. 4 (Radius and Maumenee). Left eye of same patient as in Figure 3. Note normal optic nerve with large physiologic cupping.

tic atrophy with a cup: disk ratio of nearly 0.9 (Fig. 3). Intraocular pressures were within normal limits. Examination of the left eye, however, demonstrated a similarly enlarged cup: disk ratio with an otherwise normal nerve head (Fig. 4). Even in this single instance, apparent "glaucomatous cupping" may have reflected pallor of a large but, nevertheless, normal physiologic cup.

Trauma—Thirteen patients, three with bilateral disease, were found to have optic atrophy secondary to trauma. Eleven cases involved blunt trauma to the head or orbit. In two eyes, a gunshot wound to the orbit in the absence of globe rupture resulted in subsequent atrophy of the optic nerve.

Ages ranged from 6 to 57 years with a mean age of 38 ± 15 years. Duration of optic atrophy as judged by clinical history, as well as previous examinations, ranged from one to 20 years with a mean of 4.3 ± 5.0 years. Mean cup:disk ratio in 16 eyes examined was 0.32 ± 0.13 , not

significantly different from that observed in a control population (0.30 \pm 0.12). Paired testing of contralateral uninvolved eyes in cases of unilateral disease did demonstrate a small but significant increase in cupping in eyes with optic atrophy (0.32 \pm 0.12 vs 0.28 \pm 0.11, P < .05)

Even though eyes with optic atrophy showed an increase in nerve head cupping, in no case did pathology resemble nerve head cupping characteristic of patients with glaucoma. One patient had suffered blunt trauma to the head four years before with subsequent optic atrophy. Fundus examination of the involved right eye revealed marked nerve head atrophy with what first appeared to be a large cup:disk ratio of nearly 0.9 (Fig. 5). Closer examination by slit-lamp biomicroscopy and review of stereoscopic disk photographs showed that nerve head tissue persisted across the optic disk. Though nearly transparent, this remaining tissue supported retinal vessels and effectively reduced cup size to 0.30, comparable to 0.20 recorded in the contralateral eye (Fig. 6).

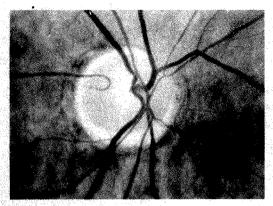


Fig. 5 (Radius and Maumenee). Markedly pale atrophic nerve head of the right eye of a patient with traumatic optic neuropathy. Slit-lamp biomicroscopy revealed cup:disk ratio of 0.3 in what appears here to be a totally cupped disk.

Demyelinating or inflammatory disease—Twenty-three cases of optic atrophy were secondary to demyelinating or inflammatory disease, or both. Twelve patients had bilateral disease. Ten patients had multiple sclerosis with peripheral neurologic deficit, whereas 13 patients had nonspecific retrobulbar neuritis.

Mean age was 44 ± 18 years, ranging from 14 to 70 years of age. Duration of symptomatology was from one to 54 years with a mean of 14 ± 14 years. Cup:disk ratios in both diseased and contralateral uninvolved eyes were 0.30 ± 0.11 and 0.30 ± 0.10 , respectively. No significant increase in nerve head cupping was seen in affected eyes when compared to either contralateral eyes or eyes of a sample population. No glaucomatous disk changes were seen. Nerve head cup:disk ratios ranged from 0.10 to 0.50.

Pressure necrosis—Optic atrophy secondary to nerve compression was seen in eight patients with either intraorbital or intracranial masses. Four patients had bilateral disease. Four patients had cranial meningiomas. Two patients had orbital tumors. One patient had an orbital abseess, and one patient had drusen of the optic nerve head.

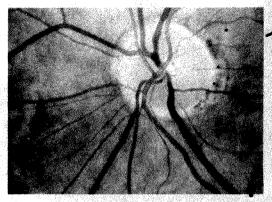


Fig. 6 (Radius and Maumenee). Normal optic nerve head in the contralateral left eve of patient described in Figure 5.

Ages ranged from 42 to 62 years, averaging 55 ± 7 years. Duration of atrophy was from two to five years. Mean cup:disk ratio of diseased eyes was 0.29 ± 0.09 ; range was from 0.10 to 0.40. This value was not significantly different from that recorded in our control population. Cases of unilateral disease were too few to make paired analyses meaningful.

In no cases were glaucomatous-like changes observed in any atrophic nerve heads. Most cases were typified by that of a 42-year-old white woman with an orbital meningioma diagnosed four years before her examination. Even after excision of the mass with section of the optic nerve, no increased cupping of the nerve head was observed (Figs. 7 and 8).

Congenital—We reviewed the cases of 27 patients with congenital optic atrophy. A variety of causes were evident, including six cases of Leber's disease; three patients had congenital syphilis, three had familial optic atrophy, and several had optic atrophy as part of the symptom complex of various congenital syndromes. All but three cases were bilaterally involved.

Ages at time of examination were from 5 to 56 years and averaged 25 ± 18 years. Mean cup:disk ratio in affected eyes was 0.35 ± 0.12 and ranged from 0.10 to 0.55. This value was significantly larger than

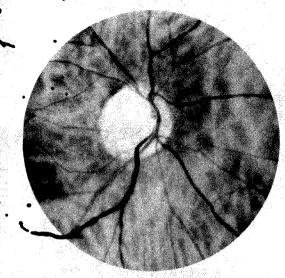


Fig. 7 (Radius and Maumenee). Right optic nerve head from 42-year-old patient two years after section of the optic nerve for excision of an orbital meningioma. Note absence of increased cupping.

that seep in a control sample. Because of sample size, comparison with contralateral eyes was not practical. In no case, however, was cupping seen that was char-

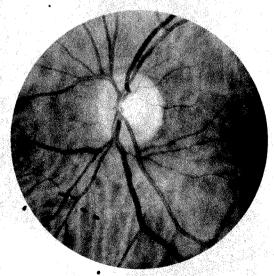


Fig. 8 (Radius and Maumenee). Optic nerve head of the contralater left eye of the patient described in Figure 7.

acteristic of glaucomatous nerve head disease.

Miscellaneous—Finally, 20 additional patients, ten with bilateral disease, were included in this series. Most of these patients were not available for examination and review consisted of examining medical records, including disk photographs. In many cases, a definite cause of optic atrophy could not be established from these records.

Ages ranged from 16 to 88 years, with a mean of 56 ± 21 years. Duration of symptomatology was 10 ± 8 years. Mean cup: disk ratio was 0.37 ± 0.22 with a range of from 0.10 to 1.00. Though larger than that seen in our control sample, differences were not statistically significant, reflecting the diversity of this sample. When compared directly to contralateral normal eyes, however, patients with unilateral disease showed significant increase in cupping in diseased eyes (Table, P < .05).

One patient of particular interest was unfortunately unavailable for repeat examination, but was included for completeness. He had been examined 20 years previously by one of us (A.E.M.) and, at that time, the patient had optic atrophy secondary to methanol ingestion. He had been described as having marked optic atrophy with 100% cupping of both optic nerve heads, Photographic material was not available for this patient. Even in this instance, however, disk changes may have reflected nerve pallor in what had previously been a large but physiologic cup or an inadequate sterescopic examination of the nerve head 20 years before this interest.

DISCUSSION

Pathogenesis of progressive cupping of the optic nerve head seen in patients with glaucomatous optic atrophy is controversial. Various authors have suggested that increased IOP precipitates vascular collapse of disk microcirculation with subsequent neuronal ischemia leading to atrophy. ⁷⁻¹³ Enlarging cup:disk ratio is presumably secondary to tissue loss.

The present study has demonstrated that in 170 eyes with optic atrophy from various causes, mean cup:disk ratio was greater than that seen in contralateral eyes as well as that of eyes of 50 diabetic patients. Significant, however, was the observation that in no case were definite glaucomatous disk changes observed. Disk asymmetry, 17,18 vertical elongation, 19.20 backward bowing of the lamina cribrosa, 9,21 notching of the neuralretinal rim,22 all of which are criteria for glaucomatous disk pathology, were lacking in our sample. Most atrophied nerve heads maintained cup: disk ratios of less than 0.40. Many nerve heads appeared entirely normal with cup:disk ratios of 0.20 or less. Even in cases demonstrating questionable nerve head changes, increased cupping may have represented atrophy in nerve heads with previously large but physiologic cupping. Similarly, subtle translucent tissue may bridge the physiologic cup. Without careful biomicroscopic inspection, these nerve heads may appear to have enlarged cup:disk "pseudo-Cases of such ratios. glaucomatous" atrophy have been described (Figs. 3-6).

Variability in estimating cup:disk ratios is well known. All patients in this report, however, were examined by one of two observers and all photographs were analyzed by both of us. When patient examination was not possible, stereoscopic photographs were used to assess physical borders of the optic cup and slide projections were used for actual measurements. These precautions should at least allow reasonable comparison of data from diseased and normal eyes. Although control patients were not matched with respect to age or sex distribution, these factors prob-

ably do not materially affect changes in cup:disk ratio.¹⁷ Indications suggest that genetic factors predominate in determining cup:disk ratio.^{17,28} It seems justifiable, therefore, to compare these two groups of eyes in this study.

None of these considerations, however, diminishes the singular observation that of 170 eyes with optic atrophy of various causes, few if any nerve heads demonstrated characteristic glaucomatous pathology. Many authors have described the difficulties inherent in distinguishing glaucomatous disk pathology from the normal optic nerve. 18,20,22,24 Similarly, it may be argued that follow up in many of these cases may have been inadequate to document significant degrees of increased cupping. On the other hand, these considerations apply to diagnosis and differentiation of glaucomatous disk changes at the earliest stage of disease process. All of these patients had well established optic atrophy with stable vision and field defects. If mechanisms of atrophy were similar in these patients suffering ischemic, compressive, or inflammatory optic neuropathies as that in patients with elevated IOP, then at least selected cases would be expected to demonstrate glaucomatous cupping of the optic nerve head. This assumption has not been borne out by this study.

Glaucomatous atrophy does not appear to result from mechanisms similar to those operative in other varieties of optic atrophy. The largest increase in cupping was seen in patients with congenital or miscellaneous forms of atrophy. In a group of 26 eyes with ischemic optic neuropathy or vascular occlusion, no significant increase in nerve head cupping was observed. Ischemic optic neuropathy would appear to be most closely analogous to microvascular collapse proposed in response to elevated pressures in glaucoma. But even in these cases of neuropa-

thy, no glaucomatous cupping was observed. Different vascular anatomy may be involved in ischemic disk disease and open-angle glaucoma. However, various anatomic studies have failed to demonstrate areas of disk vasculature specifically vulnerable to increased IOPs. 5,16

A small increase in cup:disk ratio might be expected when nerve heads undergo loss of axons in response to various noxious stimuli. Extensive cupping with backward bowing of the lamina cribrosa, however, is apparent only in glaucomatomethology is unique to increased IOP, different basic mechanisms must be responsible for disk changes characteristic of glaucoma. Perhaps the time course of the ischemic insult may be important. Glial cells, particularly resistant to acute pressure increase, may be susceptible to chronic increased pressures and secondary ischemia: Yeuronal atrophy may be secondary to loss of this neural tissue.25 On the other hand, direct mechanical compression with increased IOP may damage glial or neuronal elements directly.

Nevertheless, there is a fundamental difference in optic atrophy seen after a variety of insults to the optic nerve, including ischemia, compared to that seen in glaucoma. Consequently, it is unlikely that glaucomatous neuropathy can be a direct consequence of neuronal hypoxia secondary to compromised ocular perfusion pressures.

SUMMARY

We reviewed 170 eyes of 112 patients with optic atrophy from various causes. Special attention was directed towards measured cup:disk ratios as well as presence of glaucomatous-like cupping of the optic nerve head. We observed a small but significant increase in nerve head cupping in eyes with optic atrophy when compared to contralateral eyes, as well as

to eyes of 50 diabetic patients. No characteristic glaucomatous disk changes were documented. We evaluated these findings with respect to possible causes of glaucomatous disk and field changes.

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OPHTHALMIC MINIATURE

We believe that objects are red, blue, and so on, not solely because there is a vast majority that concur in the judgment. Their verified subjective sensations reveal more of reality than the sensations of color-blind subjects, and this is because they are natively endowed with a greater competence in seeing. Competence, and not majority opinion, is decisive.

Jacques Barzun and Henry F. Graff, *The Modern Researcher* New York, Harcourt Brace Jovanovich, 1977

SYSTEMIC HYPOTENSION AND GLAUCOMATOUS CHANGES

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The role of optic nerve ischemia in the development of glaucomatous disk and field changes remains uncertain.1-3 Proponents of the "ischemic theory" frequently cite the occurrence of glaucomatous field changes and optic nerve excavation in shock patients with normal intraocular pressures (IOP) as evidence of the importance of optic nerve vascular perfusion in the pathogenesis of glaucoma. Schwartz4 suggested that loss of visual function may be a frequent occurrence in shock patients, and that lowering the IOP in these patients may be worthwhile treatment. The development of glaucomatous changes in shock patients has not, however, been documented in a prospective fashion. This study was undertaken to determine whether we could demonstrate glaucomatous disk or field changes in a group of patients who had survived an episode of systemic hypotension with poor peripheral tissue perfusion.

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SUBJECTS AND METHODS

During an 18-month period (October 1975 to March 1977) we examined patients who had survived an episode of shock. Patients were primarily recruited from intensive care units. For inclusion in this study, a patient had to have survived at least one episode of hypotension with poor tissue perfusion lasting a minimum of one hour. Hypotension was defined arbitrarily as a decrease in systolic blood pressure to 80 mm Hg or lower in a previously normotensive patient, or a decrease of more than 50 mm Hg in a hypertensive patient. Concomitant falls in diastolic blood pressure were present in all patients as well. All patients manifested symptoms of shock, including dulled sensorium, pallor, cold clammy skin, and a rapid, weak pulse. We exluded patients if the duration or severity of shock could not be adequately documented, or if they could not cooperate for detailed visual field testing. In all, we examined 18 patients who fit these crite-

All patients had at least one complete ocular examination, including measurement of best corrected visual acuity, slit-lamp biomicroscopy, applanation tonometry, and direct and indirect ophthalmoscopy. Visual fields were determined by Goldmann perimetry (15 patients) or tangent screen testing (three patients) by

technicians experienced in the field of glaucoma. The size of the optic nerve cup was determined by direct ophthalmoscopy and ophthalmoscopic contact lens examination and documented by stereophotography. The horizontal cup:disk ratio of each eye was estimated by two observers unfamiliar with the patient's clinical history, and the mean value for each eye was recorded. Many patients had repeated field examinations and disk evaluations during this period of time.

RESULTS

These patients had 26 distinct episodes of shock; 12 patients had a single episode, five had two episodes, and one patient (Case 10) had four episodes (Table). The cause of the shock was gastrointestinal bleeding or pancreatitis in nine episodes, myocardial infarction or cardiomyopathy in 12, sepsis in three, and drug-related in two. The mean age of patients at the time of the episode of shock was 51 years (range, 2 to 84 years) and the interval between the shock and the first ocular examination ranged from one week to 17 years. The duration of the shock varied from one hour to three days.

As an indication of the severity of the shock, complications were frequently seen in other organ systems. These included renal tubular necrosis (Case 6), temporary renal decompensation (Cases 3, 4, 8, 12, 14, and 17), permanent cerebral anoxic damage (Case 13), transient cerebral anoxia (Cases 17 and 18), and gangrene of an extremity (Case 6). At the time of the initial eye examination, four of the 18 patients were determined to have ocular hypertension (IOPs greater than 19 mm Hg). However, only one patient was known to have had ocular hypertension at the time of the episode of shock (Case 10). One patient (Case 12) was in the midst of a documented bilateral elevation of IOP after cataract extractions with the use of

 α -chymotrypsin. Five days before the shock episode, his IOPs were R.E.: 43.4 mm Hg, and L.E.: 37.2 mm Hg. No pressures were determined while the patient was in shock. One patient (Case 14) went into a state of shock one hour after unilateral cataract extraction with the use of α -chymotrypsin, but his IOPs were not measured. The remainder of the patients had normal intraocular pressures.

We performed visual field examinations on 35 eyes of the 18 patients. One patient (Case 14) had an old, central retinal venous occlusion in the left eye, this eye was thus excluded from study. Thirty-one eyes had normal visual fields. The remaining four showed a peripheral scotoma from an old chorioretinal scar (Case 1), a central scotoma from senile macular degeneration (Case 9), an enlarged blind spot from old peripapillary presumed ocular histoplasmosis (Case 17), and a non-specific baring of the blind spot (Case 12).

Results of disk examinations were normal in all 35 eyes. One patient (Case 11) had significant asymmetry of the disks; cup:disk ratio was R.E.: 0.3, and L.E.: 0.1. The disks were this way one week after the episode of shock and remained unchanged.

CASE REPORTS

Case 10-A 53-year-old man had a long history of moderate hypertension and type IV hyperlipoproteinemia. He was also a heavy smoker. In 1972 the patient had a myocardial infarction with ventricular tachycardia, cardiac arrest, and congestive heart failure. In 1973 he had an episode of pulmonary embolism without shock. In May 1974 he had a second myocardial infarction with ventricular tachycardia. His blood pressure was unrecordable for two hours. A cardioverter was then used to induce a normal sinus rhythm. In August 1974 he had a coronary-saphenous vein bypass with excision of a ventricular aneurysm. Two weeks later, and again four months later, he had myocardial infarctions with cardiac arrest and episodes of shock. From 1972 to 1976 his intraocular pressures between episodes were in the low 20s. No pressures were measured during the episodes of shock. Ocular examination on Feb. 5, 1977, showed normal visual acuity, Goldmann fields, and disks. The cup:disk

TABLE

OPHTHALMOLOGIC EXAMINATIONS IN SHOCK PATIENTS

Case No.	Age at First Episode of Shock (yrs)	Sex	Cause of Shock ' No. of Separate Episodes	Durations of Shock	Interval Getween Shock & Eye Examination	Fields	Cup: Disk Ratio	. IOP at Time of Examination (mm Hg)
V	70	Σ	Myocardial infarction	12 hrs	4mos	Peripheral scotoma LE	.2 RE .1 LE	RE 10 LE 11
	<u>26</u>	E.	Septic abortion	6 hrs	2 wks	Normal (G)	.2 Both eyes	RE 11
	44	Z	Alcoholic cardiopathy	10 hrs	2 wks	Normal (G)	.4 Both eyes	LE 12 RE 12
	25	Z	(1)Gastrointestinal bleeding	4 hrs	l mo	Normal (G)	.4 RE .3 LE	LE 14 RE 16 LE 14
	2	Z	(2)Vancreatitis (1)Myocardial infarction (2)Gastrointestinal	3 hrs 3 hrs	2 wks 2 wks 8 days	Normal (G)	ARE 2 LE	
	45 42	μΣ	Dieeding Myocardial infarction Gastrointestinal bleed-	hrs. 5 hrs	1 yr 1 wk	Normal (G) Normal (G)	.3 Both eyes	Both eyes 10 Both eyes 12
	7 8	Z	Ing Septic shock (ascend- ing chalangitic)	6 hrs	1 yr 2 mos	Normal (G)	.1 Both eyes	RE 214
	99	Σ	ing cholangins) (1)Gastrointestinal bleeding (2)Gastrointestinal bleeding	hrs Uncertain	8 yrs 2 yrs	Central scotoma, RE (G)	.3 RE .2 LE	LE 20† Both eyes 13
a	3	Z	(1) Myocardial infarction (2) Myocardial infarction (3) Myocardial infarction (4) Ventricular fibril-	Uncertain 2 hrs 1/2 hr 1/2 hr	5 yrs 3 yrs 2 ^{1/2} yrs 2 yrs	Normal (G)	.3 RE 4 LE	Both eyes 21
	%	L ,	Escherichia coli Sepsis	8 hrs	wk, wks	Normal (T)	.3 RE .1 LE	Both eyes 7
	8	3	(1)Cardiopulmonary arrest 18 hrs after cataract extrac- tion (2nd eye) (2)Gastrointestinal bleed- 4 hrs ing 5 days later	18 hrs 4 hrs	1 yr	Baring of blind spot, RE (G)	.1 Both eyes	Both eyes 23
	21	×	Myocardial infarction	14.	1 24	Normal (G)	.2 Both eyes RE 12	RE 12

Both eyes 13	RE 13 LE 12	Both eyes 12		RE 21 LE 20	Both eyes 13
.4 RE (LE‡)	.4 Both eyes	,3 RE ,4 LE) RE	S RE
Normal, RE • (LE‡)	Normal (T)	Normal (G)		Enlarged blind snot L.F. (G)	Normal (T)
1 wk. 7 wks	L5 yr	2mos	7 wks	11 yrs	17 yrs
3 days	Ė	3 hrs	4 hrs	5 hrs	hrs
Myocardial infarction after cataract ex-	traction Cardiopulmonary ar- rest following bar-	biturate (1)Gastrointestinal bleeding	(2)Gastrointestinal	bleeding Myocardial infarction	Febrile seizures, drug reaction
Z	Z	Σ		Ł	Z
19	47	23		29	તા
4	10	9		r.	œ

*G indicates Goldmann field; T, tangent screen.
† Old central retinal venous occlusion.
† Receiving 2% pilocarpine four times a day at time of examination and episode of shock.

ratios were R.E.: 0.3, and L.E.: 0.4. These findings were unchanged from 1972.

Case 13-A 51-year-old man was in good health until April 1, 1976, when he suddenly fell forward with a loss of consciousness. Cardiopulmonary resuscitation was begun 30 minutes later. Approximately one hour after his initial collapse he was seen at a local hospital emergency room. An electrocardiogram disclosed alternating periods of ventricular tachycardia and ventricular fibrillation. The blood pressure was 88/46 mm Hg; several minutes later it was 90/20 mm Hg. He then underwent defibrillation. His blood pressure promptly increased to 170/ 78 mm Hg. The arterial blood pH was 7.2. The patient remained in a coma for several days. His electroencephalogram showed diffuse anoxic encephalopathy. However, during the next several weeks he showed a gradual response to external stimuli. The clinical course was complicated by pneumonia on hospital day 38, but he was discharged four months after admission with a residual right hemiparesis and some impairment of his mental function. His electroencephalogram remained abnormal. Ocular examination on Feb. 8, 1977, showed a visual acuity of 6/7.5 (20/25) in both eves. External examination and slit-lamp biomicroscopy were normal. Applanation pressures were R.E.: 12 mm Hg; and L.E.: 14 mm Hg. Goldmann fields and disks were normal.

DISCUSSION

Although acute blood loss can occasionally cause an optic neuropathy,5 the occurrence of glaucomatous disk and field changes after shock has not been documented. Drance and co-workers6 compared 45 patients with low-tension glaucoma with 45 normal and 45 ocular hypertensive subjects matched for age and sex. Thirteen of the 45 patients with low-tension glaucoma had a past history of major hemodynamic crises, but only one of 45 ocular hypertensives and four of 45 normals had such a history. In another study,7 the same investigators found that of 38 patients with low-tension glaucoma. only one of ten patients with a history of shock had a progression of eye disease, whereas eight of 28 without a history of shock demonstrated disease progression. While these studies suggest that shock is a cause in some cases of low-tension glaucoma, these data are retrospective and subject to difficulties in selecting comparable controls, as well as other potential

biases. Careful analysis of the date from another large series of cases of low-tension glaucoma shows that only three of 45 patients had a history of shock.

In our series of 26 episodes of severe shock in 18 patients, we were unable to demonstrate the development of any cases of glaucomatous disk or field changes. While we cannot conclude that this does not occur, it is uncommon. It would not be worthwhile to perform visual examinations on all shock patients or to systematically lower intraocular pressures in these patients. On the other hand, any shock patient with visual complaints should certainly have an ophthalmologic examination.

Although IOPs were not measured in our patients during the episodes of shock, it is likely that IOP falls during periods of severe hypotension because of decreased aqueous humor formation. This fall in IOP might "protect" the optic nerve from possible ischemic effects of the IOP. There would be a decrease in the normally high "tissue pressure" (IOP) within the eye. This protective effect initially made us question the occurrence of glaucomatous optic nerve changes caused by systemic hypotension.

The importance of systemic and ophthalmic artery blood pressures in the development of glaucoma remains controversial. It is frequently stated that a rapid progression of glaucoma occurs when the blood pressure of glaucoma patients with systemic hypertension is lowered. Actually, only a few such cases have been reported. 11-13 Considering the widespread use of potent systemic antihypertensive agents in millions of patients throughout the world, these few cases certainly do not prove this occurrence. It is also frequently stated that patients with low systemic or ophthalmic artery blood pressures are more prone to develop glaucomatous changes at a given level of IOP.11-14 Yet many patients with glauco-

ma or low-tension glaucoma have normal or increased blood pressures.^{9,15} We have observed a group of five patients with ocular hypertension, bilateral severe carotid artery disease, and low ophthalmic artery pressures for three to 12 years without noting the development of disk or field changes (L.M.I., unpublished data). Because important unknown susceptibility factors exist in the development of glaucomatous disk and field changes, the importance of systemic or ophthalmic ar- tery blood pressures can be demonstrated only by comparing large series of patients with similar IOPs who differ only in their systemic or ophthalmic artery blood pressure. This study has so far not been performed.

SUMMARY

We undertook a prospective study to determine the frequency of development of glaucomatous disk or field changes in a series of patients who had survived at least one episode of shock with poor peripheral tissue perfusion. In 18 patients with 26 distinct episodes of shock we were unable to demonstrate any glaucomatous disk or field changes.

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PATHOPHYSIOLOGY AND ELECTRON MICROSCOPY OF MELANOMALYTIC GLAUCOMA

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A form of secondary glaucoma resulting from trabecular meshwork entrapment of macrophages laden with phagocytized pigment from posterior segment melanomas has been described by light microscopy as melanomalytic glaucoma.1 We present the results of quantitative aqueous perfusion and transmission and scanning electron microscopy of the trabecular meshwork of an eye enucleated from a patient with a clinical picture apparently fulfilling the diagnostic criteria for this unusual entity. Clinically, the eye contained a malignant melanoma of the ciliary body and had known secondary glaucoma for approximately two weeks. Notable clinical features included an intraocular pressure of 45 mm Hg and a flat tonographic tracing. Numerous pigmented bodies slightly larger than normal uveal pigment granules were present in both the aqueous and vitreous humors. The anterior chamber angle was uniformly wide open, and a dense, black, pigmented band evenly covered the filtration portion of the trabecular meshwork (Fig. 1).

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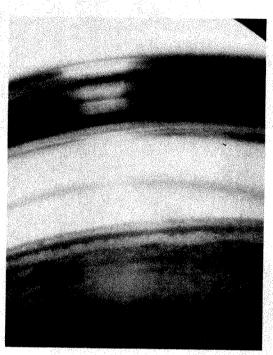


Fig. 1 (Van Buskirk and Leure-duPree). Gonioscopic view of right eye showing dense pigmentation of the filtration portion of trabecular meshwork.

MATERIAL AND METHODS

A 23-gauge needle connected to a three-way stopcock was inserted into the anterior chamber within 20 minutes of enucleation of the affected right globe. After aspiration of approximately 0.2 µl of aqueous humor from the anterior chamber via one branch of the stopcock, the eye was connected via the other branch to a quantitative constant pressure perfusion apparatus, which restored the volume of the anterior chamber. The needle was then advanced carefully through the pupil into the posterior chamber, so as not to injure the anterior surface of the

lens. Aqueous flow was measured for one hour at 15 mm Hg and for an additional hour at 50 mm Hg. The aqueous solution, Hank's balanced salt solution, was replaced by 3% glutaraldehyde in 0.1M sodium cacodylate buffer, pH 7.3, at the same intraocular pressure. The globe was opened and approximately 0.5 ml of vitreous was aspirated. The calotte containing the tumor was subjected to conventional paraffin embedding, stained with hematoxylin and eosin, and examined by light microscopy.

The aqueous and the vitreous aspirates were suspended in agar and centrifuged. The agar button containing the sediment was postfixed in 1% osmium tetroxide in 0.1M sodium cacodylate buffer dehydrated in a graded series of alcohols and

embedded in Epon 812.

Following fixation in situ, a curved strip of tissue containing the anterior chamber angle was excised from the calotte opposite the tumor and divided into three equal portions, each approximately 1.5 mm wide by 4 mm long, containing trabecular meshwork, iris root, and anterior ciliary body. The tissues were rinsed several times in 0.2M sodium cacodylate buffer containing 2% sucrose and then postfixed in 1% cacodylate-buffered osmium tetroxide. Following osmication two of the strips were dehydrated in a graded series of ethanol and embedded in Epon 812. Thick sections (1 to 2 µm) were stained with methylene blue azure and viewed with the light microscope.2 Ultrathin sections were cut on an ultramicrotome, mounted on uncoated copper grids, and stained with alcoholic uranyl acetate and lead citrate.3 Sections were examined in an electron microscope.

After fixation the third strip was prepared for scanning electron microscopy. It was dehydrated with ethanol, and treated with osmium-thiocarbohydrozineosmium (OTO) as described by Munger and Mumaw.⁴ Following the OTO treatment the tissue was dehydrated with ethanol and then placed in graded ethanol/ Freon 113 solution until it was in 100% Freon 113. The tissue was then critical point dried in a critical point dryer with Freon 113 as the drying agent, and coated with a thin (~5 nm) coat of gold/palladium with an ion sputtering apparatus. The tissue was examined in scanning electron microscopy at accelerating voltages in the range of 16 to 18 kV.

RESULTS

Quantitative perfusion—Facility of outflow measured 0.06 µl/min/mm Hg at 15 mm Hg intraocular pressure and 0.04 µl/min/mm Hg at 50 mm Hg intraocular pressure.

Aqueous and citreous aspirates—The cells in the aspirated aqueous humor had the characteristic morphology of macrophages (Fig. 2). They contained innumerable melanin pigment granules. Some macrophages were intact while others.

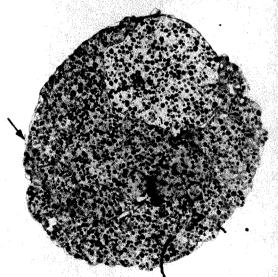
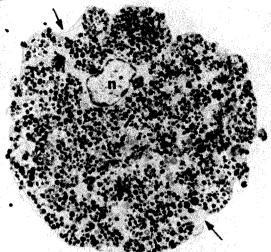


Fig 2 (Van Buskirk and Leute-duPree). Electron micrograph of a typical cell in the aqueous aspirate. Cell cytoplasm is laden with pigment granules, and filopodial processes project from the cell surface (arrow) (× 2,200).



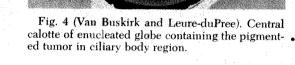


Fig 3 (Van Buskirk and Leure-duPree). Electron micrograph of a typical cell in the vitreous aspirate. Nucleus (n) is eccentrically located. Numerous pigment granules in cytoplasm. Filopodial processes are present (arrows) (× 2,200).

were in various stages of degeneration. A small amount of free pigment, also, was seen in the aqueous humor sample. The cells (Fig. 3) in the vitreous were similar to those in the aqueous consisting of macrophages in various stages of degeneration. No cells in either the vitreous or the aqueous were identified as tumor cells.

Light microscopy—A centrally necrotic, mixed spindle A and spindle B malignant melanoma was observed in the ciliary body of the calotte containing the tumor (Figs. 4 and 5).

The excised strips of anterior chamber angle appeared densely and uniformily pigmented. The intertrabecular spaces of the trabecular meshwork appeared obliterated in many areas by large attenuated cells that resembled macrophages (Fig. 6). In some areas, these cells insinuated between two to three trabecular lamellae, appearing thereby, to obstruct communication between the intertrabecular spaces.

Electron microscopy—The trabecular endothelial cells that formed a continu-

ous monolayer were located in their usual position, juxtaposed to the trabecular lamellae. In addition to the usual complement of cytoplasmic organelles, many endothelial cells contained pigment granules. Microvillous processes (4 to 8 µm in length) from these cells projected into the intertrabecular spaces (Fig. 7). Some endothelial cells were in various stages of detachment from the trabecular lamellae. while others were free in the intertrabecular spaces (Fig. 8). The integrity of the intercellular junctions between adjoining endothelial cells was often maintained (Fig. 8); however, in other areas the junetions were completely disrupted and separated.

The intertrabecular spaces were often filled with cells as large as 50 to 60 μm and the morphological features was characteristic of macrophages (Fig. 9). These cells were free within the intertrabecular spaces, and they could be distinguished from the detached trabecular endothelial cells. Their plasma membranes were irregular and often arranged as microvilli or pseudopodia. The nuclei of these cells were usually eccentrically situated and in close association with the Golgi complex. These pigment-laden macrophages also contained moderate amounts of mito-

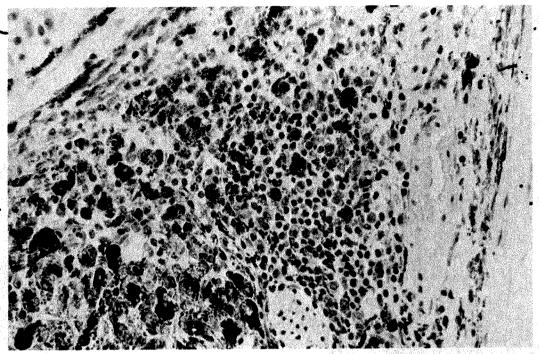


Fig. 5 (Van Buskirk and Leure-duPree). Light micrograph of the marginal region of ciliary body melanoma of mixed spindle A-spindle B type (\times 400).

chondria, ribosomes, rough-surfaced endoplasmic reticulum, lipid droplets, and a variable amount of lysosome-like structures and heterophagic vacuoles. However, smooth-surfaced endoplasmic reticulum and polysomes were rarely observed.

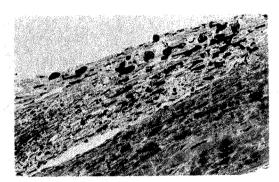


Fig. 6 (Van Buskirk and Leure-duPree). Eponembedded light micrograph of thick section of trabecular meshwork and Schlemm's canal showing multiple intertrabecular macrophages (× 250).

Microtubules were generally located in the peripheral cytoplasm.

Some cells were intermediate between "endothelial macrophages" and "histocytic macrophages," but the differentiation could not always be made with certainty. However, none of these cells were observed to have morphological features which would specifically identify them as tumor cells.

Scanning electron microscopy—Scanning electron microscopy confirmed the observation obtained from light and electron microscopy (Figs. 10 and 11). The trabecular endothelium was generally flat, with an occasiona nuclear bulge. These cells often projected processes into the intertrabecular spaces. Macrophages were observed enmeshed in the anterior chamber surface of the trabecular meshwork and in the intertrabecular spaces;

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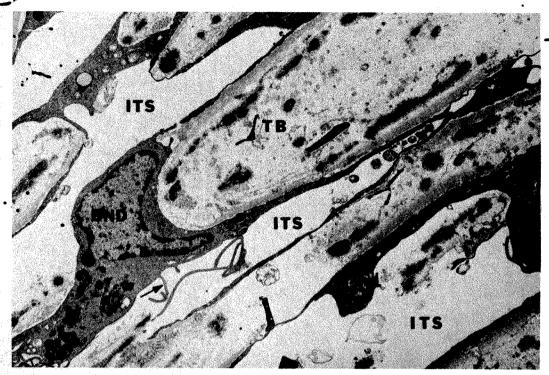
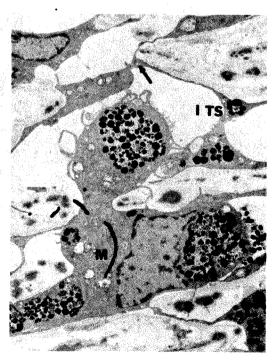


Fig. 7 (Van Buskirk and Leure-duPree). Electron micrograph of trabecular meshwork. Connective tissue core of the trabecular beam (TB) contains collagen fibrils. Trabecular endothelial cells (END) are filled with pigment granules and project slender microvillous process (arrow) into the intertrabecular spaces (ITS) (× 4.200).



their surfaces had numerous flange-like processes, giving a ruffled or irregular appearance.

Discussion

Light microscopic examination of melanomalytic glaucoma, as described by Yanoff and Scheie,¹ revealed multiple pigment-laden macrophages in the anterior chamber angle region, leading them torelate this entity to other glaucomas that have been presumed to be macrophageinduced. In the present case of melano-

Fig. 8 (Van Buskirk and Leure-duPree). Electron micrograph of trabecular meshwork. Endothelial macrophage (M) with filiform cytoplasmic extensions project into and fill the intertrabecular spaces (ITS). Macrophage contains vacuoles and residual bodies laden with melanin granules. Some cell-tocell junctions (arrow) are maintained (× 3,600).



Fig. 9 (Van Buskirk and Leure-duPree). Electron micrograph of macrophage within the intertrabectlar space. Cells have pseudopodia (arrows) and the cytoplasm contains residual bodies filled with melanin granules (× 6,000).

malytic glaucoma, the cells enmeshed the intertrabecular spaces appeared to be exclusively phagocytic in nature. We observed evidence of phagocytic activity in a variety of cells, including seemingly normal trabecular endothelial cells, semidetached, mobile endothelial cells, and wandering macrophages. This spectrum of morphology and mobility is consistent. with previous reports of endothelial phagocytosis of erythrocytes and exfoliative material and with the evidence that trabecular meshwork endothelial cells can take on all of the phagocytic capabilities of wandering macrophages. 1,6,7 As has been suggested, 7-9 this trabecular endothelial phagocytic response to chemo-



Fig. 10 (Van Buskirk and Leure-duPree). Scanning electron micrograph of the trabecular meshwork. The meshwork is lined with spindle-shaped endothelial cells that bulge into the intertrabecular spaces. Processes from these cells cross the spaces. Macrophages with irregular or ruffled appearance are present in the intertrabecular spaces (× 300).

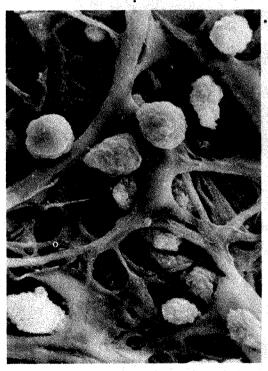


Fig. 11 (Van Buskirk and Leure-duPree). Scanning electron micrograph of the trabecular meshwork. Macrophages with ruffled appearance are visible in the intertrabecular spaces (\times 2,000).

tactic or other stimuli is similar to that of monocular reticuloendothelial surfaces. suggesting that the trabecular meshwork may act as an intraocular branch of the reticulo endothelial system. Some investigators have stressed the morphologic differentiation between histiocytic macrophages and trabecular endothelial macrophages, suggesting a distant origin for the former.5 While many of the macrophages seen in this case, especially those in the aqueous and vitreous aspirates, appeared to be wandering macrophages. the multiplicity of forms intermediate between endothelial cells and wandering macrophages makes it enticing to speculate that many of the intertrabecular macrophages may arise from the trabecular endothelium itself. Whatever their origin, it is clear from the present investigation that, while important in clearing the aq-<u>ueous humor of unwanted material, these</u> macrophages can be found in a location where one would expect them to impede the outflow of aqueous humor. In the present case, aqueous perfusion measurements on the enucleated eve before fixation clearly demonstrated abnormal obstruction to aqueous outflow compatible with the elevated intraocular pressure and low tonographic facility of outflow found clinically.

SUMMARY

We examined an eye with melanomalytic glaucoma by quantitative aqueous perfusion, light and electron microscopy. Facility of outflow was markedly diminished from normal in vivo and in vitro. The trabecular meshwork was heavily—laden on its surface and in the intertrabecular spaces with macrophages. Many of these cells appeared to be wandering macrophages, while some appeared to be detached, phagocytic trabecular endothelial cells.

ACKNOWLEDGEMENT

David Campbell, M.D., prepared the aqueous and vitreous aspirates.

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SPECTRAL TRANSMITTANCE OF INTRAOCULAR LENSES AND

RETINAL DAMAGE FROM INTENSE LIGHT SOURCES

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The special transmittance of preretinal ocular media determines the amount of radiation that reaches the retina from a light source. In the visible and nearinfrared regions of the spectrum, there is little difference between the spectral transmittances of the individual ocular media; and a substantial amount of the light incident on the cornea reaches the retina.1.2 In the near-ultraviolet region of the spectrum, however, the spectral transmittance of the crystalline lens differs markedly from that of other ocular media.1 While the other ocular media remain largely transparent, the lens transmits little incident light and acts as an effective protective filter for the retina.

A variety of intense near-ultraviolet light sources are now available; the sensitivity of the intact eye to these sources has been previously reported.3-5 Because the aphakic eye does not have the protection of the crystalline lens, it is more susceptible than the normal eye to thermal retinal damage from the absorption of light in the retinal pigment epithelium. To determine the potential sensitivity of the pseudophakic eye to intense near-ultraviolet sources, the spectral transmittance of the intraocular lens must be known. In this study I used measurements of intraocular lens transmittances to analyze the susceptibility of the pseudophakic eye to retinal damage from intense light sources.

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METHODS

Polymethylmethacrylate has been used almost exclusively in the fabrication of intraocular lenses.6 I used a Beckman DB-G double-beam grating spectrophotometer to measure spectral transmittance from 300 nm to 700 nm for clear polymethylmethacrylate intraocular lenses of the iris-suture, iris-plane, and anterior chamber types. Cuvettes were fabricated for mounting the acrylic specimens in a sample beam masked by an aperture smaller than the lens to be tested. An identical aperture was placed in the reference beam. Transmittance was recorded as the ratio of the intensities of the sample and reference beams at the photomultiplier of the spectrophotometer. I recorded results of measurements, as well as the spectral transmittances of the cornea and crystalline lens as reported by Boettner and Wolter¹ and the infrared transmittance of clear polymethylmethacrylate as presented by the United States of America Standards Institute7 (Figure).

RESULTS

The lowest wavelength at which ultraviolet spectral transmittance is significant is approximately 300 nm for cornea (also for aqueous humor and vitreous humor¹), 330 nm for polymethylmethacrylate intraocular lens, and 400 nm for crystalline lens (Figure). Below 300 nm, torneal transmittance for ultraviolet light is negligible and the retina is shielded. Above 400 nm, polymethylmethacrylate lenses and preretinal ocular media are largely transparent, and a majority of incident radiation reaches the retina. Between 300

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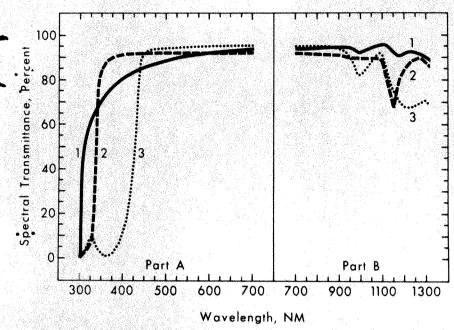


Figure (Mainster). A, Near-ultraviolet and visible transmittance measured for clear polymethylmethacrylate intraocular lenses (curve 2). No significant differences are present between the spectral transmittance characteristics of iris-suture, iris plane, and anterior chamber lenses. For comparison, the data of Boettner and Wolter¹ are included for the spectral transmittance of cornea (curve 1) and crystalline lens (curve 3). B, near-infrared transmittance for cornea (curve 1) and crystalline lens (curve 3), as well as for clear polymethylmethacrylate (curve 2).

nm and 400 nm, the cornea (as well as the aqueous humor and vitreous humor) is largely transparent and the amount of light reaching the retina depends on lenticular spectral characteristics. In the normal eye, the crystalline lens is effectively opaque in this range of wavelengths and the retina is shielded. In the pseudophakic eye, the retina has normal protection only below 330 nm because the polymethylmethacrylate lens has appreciable transmittance at longer wavelengths.

The clear polymethylmethacrylate intraocular lenses tested in this study were fabricated from Plexiglass polymethylmethacrylate (Rohm and Haas Corporation.)⁸ Perspex CQ (Imperial Chemical Industries) is a second polymethylmethacrylate commonly used in intraocular lens construction, but has appreciable transmittance at wavelengths as low as 280 nm

(at 300 nm the transmittance is still approximately 80%). Thus, the ultraviolet radiation protection of the pseudophakic eye with a Perspex CQ implant is essentially the same as that of an aphakic eye, with the cornea providing the lower wavelength limit for ultraviolet absorption.

DISCUSSION

In the range of light exposures that are neither long enough to produce photic retinopathy, 10,11 nor short and intense enough to produce acoustic transients and shock waves in the retina, 12 retinal damage from intense light sources such as continuous wave and pulsed lasers (non-q-switched) is caused primarily by light absorption in the retinal pigment epithelium and subsequent temperature increases in adjacent tissues. 13,14 Since

absorption coefficients for the pigment epithelium are greater in the near-ultraviolet than they are in the visible, 2.13.15 thermal damage will occur if sufficient near-ultraviolet light reaches the retina. The occurrence of this damage, even in eyes with intact crystalline lenses, has been demonstrated in experiments with the krypton-ion and argon-ion ultraviolet lasers.3

The data in the figure permit the following quantitative conclusions to be drawn regarding the sensitivity of the pseudophakic eye to intense ultraviolet radiation:

- 1. Because the amount of nearultraviolet light reaching the retina depends on the spectral transmittance of the lens, retinal temperature increases are also proportional to lens transmittance and the relationship between thermal retinal damage in the normal eye and in the pseudophakic eye is readily established. For example, at 350 nm (krypton-ion laser) the ratio of polymethylmethacrylate lens to crystalline lens transmittance is approximately 90:3 (Figure). Thus, at this wavelength, only 3% of the source radiance required to produce a threshold retinal burn in a normal eye should be required to produce an equivalent lesion in a pseudophakic eve.
- 2. Analysis of the data (Figure) reveals no appreciable difference in the visible and near-infrared transmittances of polymethylmethacrylate and crystalline lenses. Thus, the thresholds for thermal retinal damage at a given wavelength should be similar for the pseudophakic and the intact eye in these spectral regions. The recent clinical findings of Poole and Galin¹⁶ support this conclusion.
- 3. Retinal temperature rise is proportional to thermal source strength, which in turn is proportional to the product of the pigment epithelial absorption coefficient (α_1) and the preretinal ocular transmittance (T_e) .^{2,13} Ocular transmittance

for the pseudophakic eye is proportional to ocular transmittance for the intact eye (T_e) multiplied by the ratio of the transmittance of the polymethylmethacrylate lens (T_p) to the transmittance of the crytalline lens (T_c) . Thus, for exposures in the millisecond range or shorter, the ratio of retinal temperature rise (v) in the pseudophakic eye at 500 nm to temperature rise in the pseudophakic eye at 350 nm is

$$\frac{v \text{ (at 500 nm)}}{v \text{ (at 350 nm)}} = \frac{A \text{ (at 500 nm)}}{A \text{ (at 350 nm)}}, \bullet$$
where

 $A \propto \alpha_1 \cdot T_e \cdot (T_p/T_c)^{\bullet}, {}^{\bullet}$ where T_p and T_c are given (Figure) (at 500 nm, T_p and T_c are 0.92 and 0.94, respectively; at 350 nm, T_p and T_c are 0.84 and 0.02, respectively), and where T_e and α_1 are given² (at 500 nm, T_e and α_1 are 0.45 and 900, respectively; at 350 nm, T_e and α_1 are 0.015 and 7000, respectively). Thus, only 9% of the source radiance required to produce a threshold retinal burn in a pseudophakic eye with an argon laser (at approximately 500 nm) should be • required to produce an equivalent thermal lesion in a pseudophakic eye with a krypton-ion laser (at approximately 350 nm).

Both the increased sensitivity of the pseudophakic eye to retinal damage from intense near-ultraviolet light sources and the chromatic aberration introduced by clear polymethylmethacrylate transmittance of natural background nearultraviolet radiation can be eliminated by fabricating intraocular lenses from acrylics with ultraviolet absorbing characteristics more like those of crystalline lens.⁷⁻⁹ While ultraviolet absorbing ints have been useful in spectacle lenses prescribed for aphakic eyes,17 vistal results with current clear polymethy methacrylate intraocular lenses are excellent.6 In view of the limited usage of potentially hazardous ultraviolet sources, testing a new class of acrylics is not justified by this unlikely hazard. Nonetheless, current clear polymethylmethacrylate intraocular lenses do not provide the same level of protection against near-ultraviolet light as does the original crystalline lens. This must be considered in formulating permissible exposure levels for such radiation, and in counseling pseudophakic patients about possible occupational or environmental hazards.

SUMMARY

I measured the spectral transmittance of clear polymethylmethacrylate intraocular lenses in the 300- to 700-nm range. The near-ultraviolet transmittance of the polymethylmethacrylate lens was significantly greater than that of the crystalline lens. Therefore, the pseudophakic eye is more susceptible to retinal damage from intense near-ultraviolet light sources than the normal eye. Retinal thermal response of the pseudophakic eye was compared with that of the normal eye for nearultraviolet radiation, and retinal thermal response to near-ultraviolet radiation was compared with that to visible light for the pseudophakic eye. Additionally, because there was no significant difference between polymethylmethacrylate and crystalline lens in visible and near-infrared transmittance, thresholds for thermal retinal damage at a given wavelength are similar for the pseudophakic and the intact eye in these spectral regions.

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CHORIORETINAL VASCULAR ANASTOMOSES AFTER PERFORATING TRAUMA TO THE EYE

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Perforating injuries involving the choroid and retina may lead to retinal detachment, vitreous hemorrhage, or fibrovascular ingrowth. They may also heal uneventfully, sometimes even in the absence of a prophylactic cryotherapy or diathermy coagulation. A previously unreported consequence of perforating chorioretinal trauma is choroidoretinal vascular anastomosis. I observed this disease in a patient ten months after an intraocular foreign body struck the fundus and lacerated the retina and choroid.

CASE REPORT

A 32-year-old, healthy white man was hammering a truck part when he sustained a perforating injury in the right eye from a small piece of metal. Visual acuity was hand motions at two feet. In the 7 o'clock meridian of the right eye, a 2-mm long, perforating injury through the corneoscleral limbus and a peripheral iridotomy were present. The lens was clear, but visualization of the fundus was prevented by a small hyphema and extensive vitreous hemorrhage.

Orbital x-ray films disclosed a $5 \times 2 \times 1$ -mm foreign body that appeared to be within the globe. The intraocular location was confirmed by an ultrasound study, which also indicated an attached retina. One day after the injury the corneal laceration was repaired, and the vitreous hemorrhage cleared sufficiently to allow visualization of the foreign body inferotemporally, where it lay in the vitreous near the pars plana. No fundus details could be seen. The metallic object was removed with a magnet through a pars plana sclerotomy. The scleral incision was sutured and then surrounded by cryotherapy. No complications occurred during the operation or in the immediate postoperative period.

Five weeks postoperatively, the vitreous blood

had cleared sufficiently to permit visualization of the fundus. A flat, curvilinear chorioretinal scar was seen superotemporal to the macula, posterior to the equator, marking the site of initial impact against the wall of the eye by the foreign body. A vitreous traction band connected this scar with old, inferiorly located vitreous blood. During the next two months, the band spontaneously disappeared.

Follow-up examination ten months after the injury disclosed best corrected visual acuity of R.E., 6/9(20/30)-1. The anterior segment showed no signs of inflammation and, except for a well-healed corneal scar, it was completely normal. Intraocular pressure by applanation was 13 mm Hg bilaterally. Gonioscopy showed peripheral anterior synechiae at the 7 o'clock position that concealed the traumatic iridotomy. Goldmann perimetry disclosed normal peripheral fields.

Inspection of the right fundus through a dilated pupil showed the retina to be flat. The macula and disk appeared normal. Old chorioretinal scarring was present in the area of the cryoapplications (inferotemporally, between the equator and the onservata). The foreign body impact site, just superotemporal to the macula, was flat, had no surrounding hyperpigmentation, and no adherent vitreous band. A retinal arteriole near the lower pole and a venule near the upper pole of the scar were seen dipping into the chorioretinal lesion (Fig. 1).

Fluorescein angiography of the chorioretinal sear showed most of the retinal pigment epithelium to be absent; many large choroidal vessels were noted

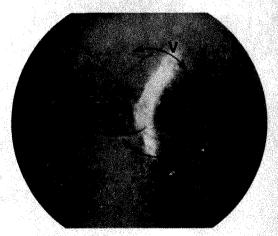


Fig. 1 (Goldberg). Chorioretinal scar ten months after perforating injury. Note deviated retinal arteriole (A) at lower pole of scar and retinal venule (V) at upper pole of scar.

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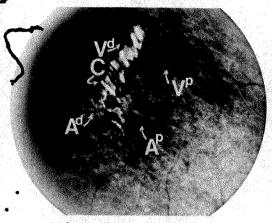
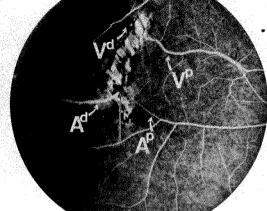
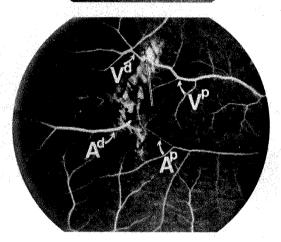


Fig. 2 (Goldberg). Angiographic sequence showing choroidal perfusion of retinal arteriole. Top left, Early arterial phase of angiogram. Note choroidal perfusion (C), early filling of distal segment of interrupted retinal arteriole (A^d), but absence of filling of its proximal segment (A^P). Neither the distal nor the proximal segment of the retinal venule is perfused yet (V^d and V^P). Top right, Early venous phase of angiogram. Note discontinuous retinal arteriole (A^d and A^P) and choroidal origin of distal segment of retinal arteriole (long arrow). The proximal portion of the retinal venule (V^P) is perfused, but the distal segment (V^d) is not. Bottom right, Late venous phase of angiogram. Note apparent discontinuity in retinal venule (long arrow).





within the scar during the choroidal phase of the dye's transit (Fig. 2). Within less than one second thereafter, the distal segment of the retinal arteriole, located at the lower pole of the chorioretinal scar, demonstrated perfusion that originated from the choroidal, rather than from the retinal, circulation. Subsequent angiographic photographs showed that the proximal and distal segments of this retinal arteriole were no longer in continuity. The retinal venule at the superior pole of the scar appeared to be interrupted within the depth of the scar, but the two-dimensional nature of the angiogram prevented absolute confirmation of this discontinuity. There was no abnormal leakage, transudation, or staining. No neovascular tissue was seen in or adjacent to the scar.

DISCUSSION

Analysis of this case indicates that the chorioretinal scar in the posterior fundus was caused by direct impact of the perforating metallic foreign body, which then bounced off the wall of the eye into the vitreous. Subsequent surgery through the pars plana was remote from the chorioretinal scar, and could not have caused it.

Fundus photographs taken ten months after the injury showed interruption of the continuity of a major retinal arteriole, the distal segment of which derived its blood supply from the choroid. Clearly, the piece of metal had lacerated the retina and choroid. As a result of the breaching of Bruch's membrane and the retinal pigment epithelium, lacerated vascular segments from the ordinarily separate vasculatures of the retina and choroid came together, and a functional anastomosis occurred during the healing process. Similar events have taken place as a result of

nontraumatic cicatrizing fundus inflammatory disease,2 as well as after excessively intense photocoagulation^{3,4} and after blunt trauma with choroidal and retinal rupture. In all of these events, the integrity of Bruch's membrane and the retinal pigment epithelium has apparently been lost, and the approximation of the damaged vasculatures of the retina and choroid has resulted in chorioretinal vascular anastomoses. In some cases, neovascular capillary growth has occurred, with subsequent subretinal and intraretinal transudation and hemorrhage.2.5 Anastomosis of large caliber retinal and choroidal vessels, however, appears to have a benign course; it is apparently not complicated by clinically significant transudative, hemorrhagic, or proliferative events.

SUMMARY

A 32-year-old white man sustained an ocular injury with a small piece of metal. He had perforation of the retina and choroid, and later developed chorioretinal

vascular anastomosis. Disabling complications, such as neovascular growth, rous transudation, or hemorrhages, did no occur within a ten-month follow-up period. Interruption of Bruch's membrase and the retinal pigment epithelium, with approximation of the normally separated vasculatures of the choroid and retina, appears necessary for such anastomoses to occur.

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OCULAR MANIFESTATIONS OF GROUP A NIEMANN-PICK DISEASE

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In 1914 Albert Niemann¹ first described an infant with Group A Niemann-Pick disease. No mention was made of any ocular abnormality. Two years later, Knox² reported clinical and pathologic findings from two sisters who resembled the infant reported by Niemann. Cherryred spots were noted in the fundi of one of the sisters, whereas the fundi of the other were not described. In 1927, on the basis of a review of pathologic material, Pick³ concluded the disease was a distinct nosologic entity and not a variant of Gaucher disease as it had been considered. In 1934, Klenk⁴ reported that the predominant lipid that accumulated in the liver and brain was sphingomye-. lin. Niemann-Pick disease is now classified into four apparent phenotypes, Groups A-D.⁵⁻⁷ In all types cholesterol accumulates, as well as sphingomyelin. In 1966 the activity of an enzyme, sphingomyelinase, was found to be markedly deficient in the tissues of patients with Groups A and B Niemann-Pick disease.7 To date, no discrete enzyme deficiency has been identified in Groups C and D.6,8,9

We report the ocular findings present in four infants with Group A Niemann-Pick disease. Retinal cherry-red spots were present in all patients, as has been described frequently in this condition.8 Corneal and lenticular changes, which have not been reported previously, were also present, suggesting widespread ocular involvement in this disorder of sphingolipid metabolism.

SUBJECTS AND METHODS

The four patients described in this report were all examined here between 1968 and 1974. All of the patients were examined by each of us and the reported ocular findings were observed by at least one other consulting ophthalmologist.

Each patient was seen during the first year of life because of abdominal enlargment resulting from hepatosplenomegaly. Bone marrow aspiration in each demonstrated foam cells. Vacuolated lymphocytes were found in the peripheral blood smears of all patients. The clinical course of each child was consistent with that expected for Group A Niemann-Pick disease, with death occurring at 17 to 32 months of age. Postmortem chemical evaluation of Cases 1 and 3 revealed increased sphingomyelin and cholesterol in the viscera and cortical gray matter. White blood cell sphingomyelinase activity was measured in Cases 3 and 4 and was found to be absent.

CASE REPORTS

Case 1—When seen at 11 months of age, this girl was not known to have visual problems or ocular abnormalities. Visual examination showed steady fixation and smooth following. Eye movements were full without nystagmus. The corneas were of normal size, but were cloudy by hand-light examination. No epithelial or endothelial defects were seen. The anterior chambers were clear and the irides were normal. The pupils were equal in size and reacted normally to light. The lenses in each eye showed a

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Reprint requests to David S. Walton, M.D., 243 Charles Street, Boston, MA 02114. brown discoloration of their anterior surface. There was also an opacity on the posterior capsule of the right lens. Both fundi showed cherry-red spots. The central red spot was dark red in each eye and the surrounding white halo extended into the perimacular region. The disks and retinal vessels were normal. The peripheral retina was not examined (Table).

Case 2-When seen at 10 months of age with hepatosplenomegaly and suspected Niemann-Pick disease, this boy had no ocular symptoms. Ocular examination revealed attentive fixation and following, normal pupillary responses to light, and intact optokinetic nystagmus. The patient's eye movements were full without nystagmus. Both corneas by hand-light and slit-lamp examination were mildly cloudy. An increase in stromal translucency could be seen on slit-lamp examination. Both lenses showed a definite granular brownish deposit on or in the anterior capsule. The fundi possessed a typical foveal cherry-red spot with marked white opacification of the parafoveal retina and slight but definite translucency of the retina throughout the posterior pole of each eye. The disks and retinal vessels were normal. We saw the patient again at 1 year of age, and detected no changes in these ocular abnormalities (Table).

Case 3—We saw this girl at 3 months of age with hepatosplenomegaly and a history of normal visual behavior. Ocular examination revealed fixation and following; pupillary reaction to light and response to an optokinetic drum were normal. The ocular movements were full without nystagmus. The corneas were diffusely cloudy because of increased stromal opacification. The corneas were not thickened, nor was there epithelial edema. The intraocular pressure was 18 mm Hg in both eyes. The anterior chambers were clear. The irides were brown and smooth. The lenses possessed a brownish dis-

coloration of their anterior surfaces. On slit-lamp examination, this lenticular opacification appeared fine and granular. It was most dense centrally, and lessened appreciably near the equator of the lens. The posterior capsule of each lens had many small, white spots located on or in this membrane. These resembled keratitic precipitates and were distributed without any order. The fundi showed cherryred spots; the central spot was dark red in color. The concentric parafoveal retinal opacity was white and seemed to spread with much less density throughout the whole posterior pole of each eye. The disks and retinal vessels were normal.

On re-examination at age 8 months and 18 months, no changes in the patient's ocular abnormalities were detected. We saw the patient for ocular examination at 28 months of age, and for the last time at 32 months of age. At these times, she showed evidence of visual deterioration with only questionable visual responsiveness. Fixation and following were absent and a bright light was tolerated without objection. The pupils responded to light only slightly. Corneal clouding persisted. On slit-lamp examination, the previously described anterior and posterior lens abnormalities appeared unchanged. Ophthalmoscopy showed the persistence of the cherry-red spots, the mild generalized retinal opacification, and normal vascularity of the optic disks (Fig. 1).

Case 4—We saw this girl at 6 months of age for air ocular examination because of hepatosplenomegaly and suspected cloudiness of the corneas. Ocular examination showed normal following movements, but unsteady fixation in the primary position changing to nystagmus on right or left gaze. The pupils responded well to light. The corneas were of normal size, but were diffusely hazy. Slit-lamp examination revealed the stromal location of the corneal opacifi-

TABLE
OCULAR FINDINGS IN PATIENTS WITH GROUP A NIEMANN-PICK DISEASE*

The second secon	Case Nos.				
	1	2	3	4	
Age at examination Visual behavior Nystagmus Pupil reaction to light	11 mos Normal None Normal	10 mos Normal None Normal	3 mos Normal None Normal	6 mos Unsteady fixation Present Normal	
Cornea Stromal opacity	4	+	+	/ 🔥 .	
Lenses Anterior capsule opacity	+	+	+	+	
Posterior capsule opacity Fundi	0	0	+	. +	
Cherry-red spot	+	→	+	+ :	
Diffuse retinal opacification Normal IOP	++	+ Not measured	+	+ +	

^{*}Present, +; absent, -.

cation. The lenses showed brown granular discoloration of their anterior surfaces. This abnormality was most dense centrally. Approximately ten scatered white spots were seen in or on the posterior capsule of each lens. They were of moderate size and again resembled the corneal precipitates of indocyclitis. The vitreous was clear. The fundi showed cherry-red spots bilaterally. The central red spot was dark red and was surrounded by a concentric white retinal opacity with an indistinct outer border. The retina beyond the macular area seemed to possess a mild opacification. The disks and retinal vessels were normal.

Repeat ocular examination when the child was 11 months old revealed persistent and increased opacification of the corneas, lenses, and retinas. The corneal opacity was diffuse throughout the full thickness of the corneal stroma. The brown anterior lens capsule opacity was more dense, and was present with equal intensity over all of the anterior surface that could be seen through dilated pupils. The small, posterior capsule lenticular opacities had greatly increased in number, but were still white and approximately the same size. The macular cherry-red spot had not changed, but the equatorial and peripheral retina seemed to show more striking opacification.

DISCUSSION

The cherry-red spot retinal lesion is a well known manifestation of Group A Niemann-Pick disease (Fig. 1). Other diseases that manifest a cherry-red spot are Tay-Sachs disease ($G_{\rm M2}$ -gangliosidosis, Type I), Sandhoff disease ($G_{\rm M2}$ -gangliosidosis, Type II), ¹¹ generalized gangliosidosis ($G_{\rm M1}$ -gangliosidosis, Type I), ¹² juvenile neuronal lipidosis with myoclonus, ^{13–15} Farber disease, ¹⁶ mucolipidosis Type I, ¹⁷ and a few other conditions. ^{18–20}

Though the cherry-red spot is considered a constant feature of Tay-Sachs disease, its incidence of Group A Niemann-Pick disease is less certain. An occurrence in 50% of such cases has been reported. Despite the prominence of this abnormality, it can be easily missed when the fundiare examined through undilated pupils in a general pediatric physical examination.

The retinal pathology in patients with Niemann-Pick disease has been described. 21-27 Abnormal lipid accumulation in the form of membranous cytoplasmic inclusion bodies is present in the

retinal ganglion cells, amacrine cells, receptor cells, and pigment epithelium. 25-27 It is characteristic of children with Group A Niemann-Pick disease to have delayed visual loss; whereas in Tay-Sachs disease. amaurosis and optic atrophy occur relatively early in the clinical course. Pathologically, this functional difference can be correlated with the preservation of ganglion cells in Niemann-Pick disease. as compared to the progressive dissolution of ganglion cells in Tay-Sachs disease. Although the cherry-red spots in these two diseases resemble each other, subtle differences exist. The central red spots in Tay-Sachs disease and Niemann-Pick disease are indistinguishable; and their darkness or brightness depends on the amount of overall choroidal pigmentation. The white ring surrounding the cherry-red spot of Niemann-Pick disease, however, is less sharply demarcated from the surrounding retina than in Tay-Sachs disease. In Niemann-Pick disease the perifoveal white opacity extends peripherally as a generalized, mild, retinal opacification in contrast to the transparency of the equatorial and perimacular retina in Tay-Sachs disease. Furthermore, in Niemann-Pick disease the white ring persists until death, while in Tay-Sachs disease it may become smaller in width after the first year of life with its outside perimeter approaching the central cherry-red spot (Figs. 2 and 3).

The observation of corneal and lenticular abnormalities in four consecutively examined infants with Group A Niemann-Pick disease suggests that these features may occur in a high percentage of such patients (Fig. 4). Concentration on the familiar retinal signs of this disease may result in neglect of anterior segment abnormalities.

The corneal abnormality present in each child was a mild opacification appearing stromal in origin and related to a diffuse increase in stromal translucency.

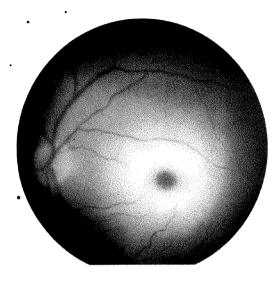


Fig. 2 (Walton, Robb, and Crocker). The fundus of a 28-month-old patient with Group A Niemann-Pick disease (Case 3). The typical cherry-red spot and surrounding retinal opacity at the macula is shown. The disk appears normal.

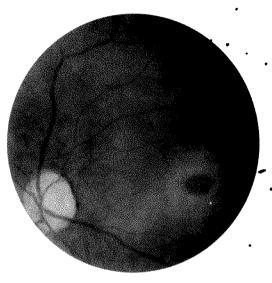


Fig. 2 Walton, Robb, and Crocker). The fundus of the left eye of a 15-month-old child with Tay-Sachs disease. Note the typical cherry-red spot and early optic atrophy.

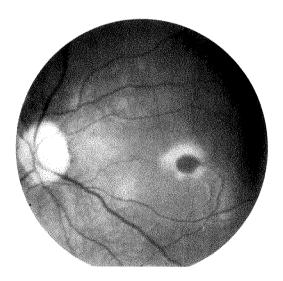


Fig. 3 (Walton, Robb, and Crocker). The left fundus of the same patient as shown in Figure 2 at 35 months of age. The loss of the white concentric parafoveal white opacity occurred over the interval of 20 months.

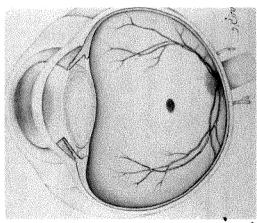


Fig. 4 (Walton, Robb, and Crocker). Drawing of the abnormalities seen in the cornea, lens, and fundus of infants with Group A Niemann-Pick disease. Note the diffuse corneal opacification, the brown granular anterior lens abnormality, the posterior capsular white spots, and the cherry-red spot abnormality at the fovea.

In no instance was this finding associated with visible corneal epithelial abnormalities, iris abnormalities, or glaucoma. The defect qualitatively resembled the corneal change seen in Hurler syndrome, but was quantitatively less marked. This abnormality in corneal transparency in Niemann-Pick disease appears to be caused by abnormal lipid accumulation in the corneal stromal and endothelial cells.25 and in the corneal epithelium.26 The morphological similarity of this corneal lipid to the microscopic abnormalities found in other body tissues has suggested that this material in the cornea is also sphingomyelin and cholesterol.25

Diffuse corneal clouding has been recognized in other metabolic illnesses including the mucopolysaccharidoses, Types I-H, I-S, IV, and VI; in G_{M1} gangliosidosis, Type I; and in the mucolipidoses. 17,28-30 Diffuse corneal clouding has been absent in conditions with only excessive sphingolipid accumulation. Lipid disorders associated with localized corneal opacities include familial lecithin-cholesterol acyl transferase deficiency,31 Fabry disease,32 Tangier disease,24 and familial hyperlipoproteinemias.24 In all of these except Fabry disease the abnormality is in the corneal stroma; in Fabry disease the defect is deep in the corneal epithelium.33 The association of corneal clouding and a retinal cherry-red spot has been previously reported in G_{M1}-gangliosidosis, Type I,¹² mucolipidosis, Type I (lipomucopolysarrharidosis),17,34 in cases reported by Goldberg¹⁹ and by Yamomoto²⁰, and in a single case of Sandhoff disease.35

The anterior and posterior lens opacification in our patients was mild and could not have caused significant visual loss. The brown granular opacification of the anterior lens was seen best with magnification and white light illumination. This abnormality was most dense axially and gave the appearance of light brown paint

sprayed on the anterior capsule. The posterior capsule opacities in Cases 3 and 4 were identical. This change was first seen in Case 3 and could have been missed during the examination of two patients (Cases 1 and 2). These numerous spots were round, flat, whitish structures reminiscent of flat keratitic inflammatory precipitates. No associated vitreous abnormality was identified.

Lens opacification or discoloration is unusual in lipid storage diseases, but has been described in phytanic acid storage, disease (Refsum disease)³⁶; in Fabry's disease³² as a posterior subcapsular sutural opacity; and with cholesterol storage in cerebrotendinous xanthomatosis.^{37,38} In both Fabry's disease and Niemann-Pick disease, abnormal accumulation of lipid has been identified pathologically in the lens epithelium.^{25,26,33}

Correction—Because of a typographical error Figure 1 was incorrectly labeled.

SUMMARY

Four infants with Group A Niemann-Pick disease had similar ocular abnormalities secondary to this systemic disease. Each child demonstrated corneal opacification, brown discoloration of the anterior lens capsule, and retinal opacification with a macular cherry-red spot. These abnormalities were seen in each child during the first year of life and appeared stable. Recognition of this combination of ocular defects facilitates early identification of patients with Group A infantile Niemann-Pick disease.

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PIGMENT EPITHELIAL PROLIFERATION IN HUMAN RETINAL DETACHMENT WITH MASSIVE PERIRETINAL PROLIFERATION

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Despite its frequent occurrence, massive periretinal proliferation, the dreaded complication of retinal detachments is a little understood disease. Analysis of experimental retinal detachments¹⁻¹⁰ has shown this to be a complication that is caused by proliferation and membrane formation of cells probably deriving from retinal pigment epithelium and from retinal glial elements.

A first histological analysis of human tissue obtained during vitrectomy was published by Constable and associates.⁴ A detailed analysis is still outstanding. In the present paper, we describe the histologic and electron microscopic findings of specimens taken from human eyes that underwent vitrectomy and preretinal membrane peeling in an attempt to treat this disease. This paper is concerned with tissue that we think derives from pigment epithelial cells. The analysis of tissue deriving from glial cells has been described.⁵

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MATERIAL AND METHODS

Thirty-nine patients with retinal detachment and massive periretinal proliferation (MPP)1 underwent vitrectomy via the pars plana with the vitreousinfusion-suction-cutter (VISC) combined with peeling of preretinal membranes.^{2,3} At surgery specimens were collected from each patient by aspiration through the VISC. A total of 21 biopsy specimens of preretinal membranes and 20 biopsy specimens of vitreous were obtained in Ringer's or lactated Ringer's solution and prepared for histologic and electron microscopic examination. The specimen was added to one or two volumes of cold 5% buffered glutaraldehyde and kept at 4° C for one day. The specimens were then centrifuged (2,400 rpm for five minutes) and the sediment postfixed in phosphatebuffered osmium tetroxide, dehydrated in a graded series of ethanol, and embedded in Epon. Thick sections (1.5 µ) for phase contrast light microscopy were stained with paraphenylenediamine. Thin sections (60 nm) were stained with uranyl acetate and lead citrate and examined under electron microscopes.

RESULTS

Cell morphology, distribution, and organization—Both the vitreal and preretinal membrane specimens showed similar cell types and great variety in cell arrangements. Some areas of a typical specimen contained predominantly isolated,

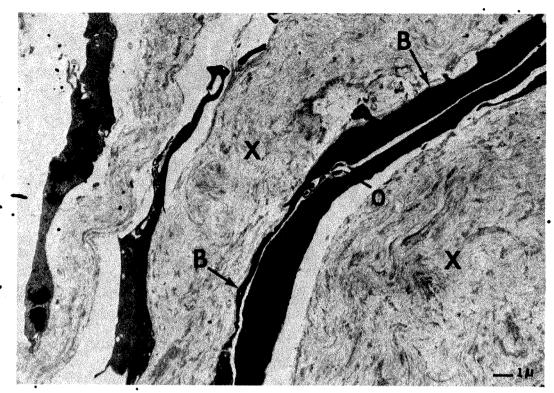


Fig. 1 (Machemer, van Horn, and Aaberg). Elongated cells surrounded by extracellular matrix consisting of callagen and granular material (X). Cells appear polarized with basal lamina (B) on one side. The space between cells (0) has no electron-dense opacities.

elongated cells embedded in large amounts of extracellular matrix composed of masses of collagen-like fibrils (13.5 to 19.1 nm in diameter) and amorphous granular material (Fig. 1). Other areas showed a predominance of clustered cells with short cell bodies and multiple cytoplasmic processes. Most of the cells were surrounded by extracellular matrix (Fig. 2). In areas of high density, the cells were polymorphous and had numerous coarse and fine cell processes that were sometimes extremely long, and interdigitated freely with processes of other cells (Fig. 3). These cells showed basal laminar formation and extensive deposits of extracellular material on the side of the cell where the cell processes were usually coarse. On the side of the fine cell processes, however, no electron-dense material was depos-

ited (Fig. 3). Since basal lamina and extracellular matrix material always occupied the opposite side of the cell from the fine cytoplasmic processes, we believed this to be an indication that the cells were polarized.

A similar polarization was seen in some cell clusters where an almost rosette-like configuration of densely packed cells was found (Fig. 4) with interdigitation of fine cell processes in the center and smooth surfaces opposing the adjacent cell. In the cell clusters, as well as elsewhere, specialized cell junctions including tight- or gap-junctions, desmosomes (Fig. 5), and sometimes even combinations suggesting junctional complexes were seen.

Intracellular characteristics—Except for the elongated cells, where the nucleus was long and oval (Fig. 1), the cells had a



Fig. 2 (Machemer, van Horn, and Aaberg). Cell clusters with irregularly shaped cells and multiple cytoplasmic processes surrounded by extracellular matrix (X), pigment granules (P), and lipid-like droplets with membranous material (L).

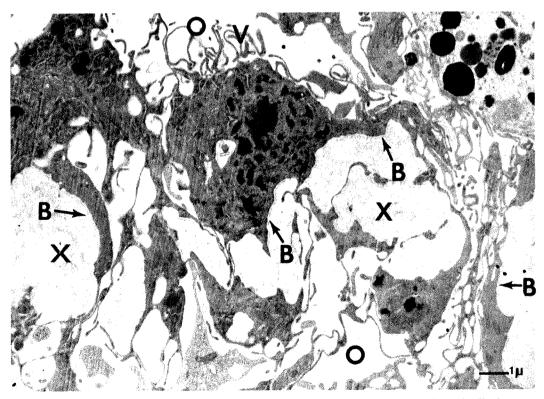


Fig. 3 (Machemer, van Horn, and Aaberg). High-density cell clusters with highly polarized cells showing basal lamina (B) and extracellular matrix (X) on one side and fine cytoplasmic processes (V) on the opposite side of the cell where the intracellular space is electron empty (0).

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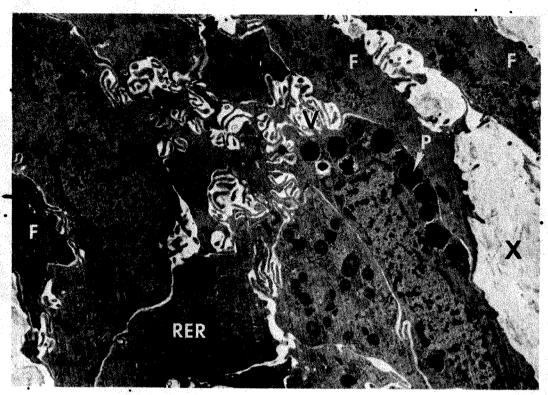


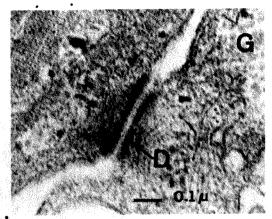
Fig. 4 (Machemer, van Horn, and Aaberg). Rosette-like configuration of densely packed cells with pigment granules (P); intracellular filaments (F) located at margin of cells; prominent rough endoplasmic reticulum (RER).

compact, sometimes invaginated nucleus. usually located in the center of the cell (Figs. 2-4). Most of their heterochromatin was marginally located. The cytoplasm usually demonstrated prominent dilated cisternae of rough endoplasmic reticulum (Figs. 4 and 6). Typical were nonmembrane bound, sometimes large accumulations of granular material (granular size, 32.5 to 42.5 nm) that represented glycogen (Figs. 5 and 6). These were commonly seen in the isolated cells, but rarely in the cell clusters. Lamellar inclusions and dense bodies (Fig. 3) were frequent. Lipid-like droplets were often located near or in the area of the granular material and contained membranous material (Fig. 2). These structures were less frequently in cell clusters than in isolated cells.

A few cells contained an abundance of intracellular filaments about 10 nm in diameter. They were usually densely packed at the margin of the cell and in cell processes in areas void of any other structures (Figs. 4–6). They often had an orderly arrangement in a fascicular pattern. Some cells contained thinner filaments, which were less well defined (Fig. 7). They appeared in bundles, showed a fine striate pattern, and had fusiform densities.

Occasionally, densely stained round or oval pigment granules were in the cells (Figs. 2, 4, and 8). Most of them were located in phagosomes and showed various degrees of disintegration. Some, however, were free and nonmembrane bound (Fig. 4).

Interspersed with the typical cells were



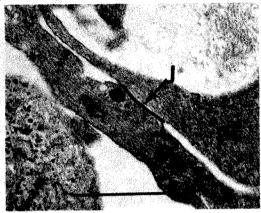


Fig. 5 (Machemer, van Horn, and Aaberg). Top, Desmosome (D) and glycogen granules (G); bottom, cell junction (J) and intracellular filaments (F).

heavily pigmented cells at various stages of vitality (Fig. 9). They contained dark, round or wedge-shaped granules and less densely stained round bodies containing homogenous material. Both granule types were frequently incorporated in phagosomes, but the granules also occurred nonmembrane bound. The granules were in various stages of degeneration, consistent with autolysis.

Relation of cells to internal limiting membrane—Six specimens contained portions of basal lamina of the retina (internal limiting membrane). The vitreal side of the lamina had a smooth surface, whereas the retinal side had an irregular contour (Fig. 8). Cells on the vitreal side of the lamina were either in direct contact

with it, or separated from it by a thin layer of fibril-containing material. The cells found on the vitreal side of the lamina had morphology, cytoplasmic organelles, and inclusions similar to those of the cells described in the vitreous (Fig. 8, top). The cells in contact with the retinal side had elaborate pseudopod-like extensions into the irregularities of the lamina. The remainder of the cell body was often separated from the lamina by interposed granular or filamentous material. Rarely, whole cell wall was seen attached to the. lamina. These cells also greatly resembled those found preretinally and in the vitreous (Fig. 8, bottom). There were remnants of retinal cells and cells containing myelin bodies, which might represent phagocytosed retinal material. None of these cells showed specialized foot plate processes or other characteristics that would mark them as normal retinal or glial cells.

DISCUSSION

Two major types of cells could be distinguished morphologically in our biopsy specimens of human MPP. There were spindle-shaped cells with long stretched nuclei and scarce cytoplasm surrounded by fibrillar material that presumably represented collagen. There were also shorter cells with irregularly shaped and indented nuclei with abundant cytoplasm, multiple thin cytoplasmic processes, and basal lamina-like substance extracellularly. These cells usually appeared in clusters or even in a rosette-like arrangement. Characteristically, these cells contained a well-developed, rough endoplasmic reticulum with wide cisternae. Glycogen was often found in abundance. Dense bodies and lipid bodies were also found. Large bundles of intracellular filaments of two types were typical: filaments 10 nm in diameter, and sometimes thinner filaments with fusiform densities of about 6 nm in diameter.

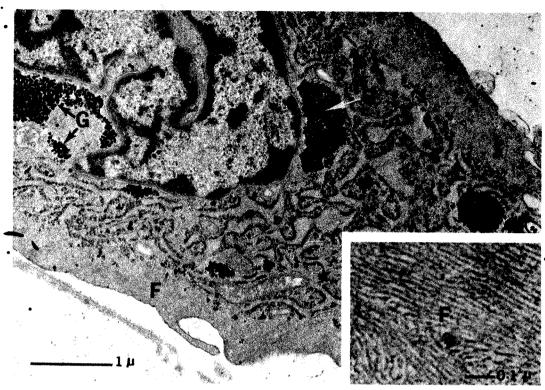
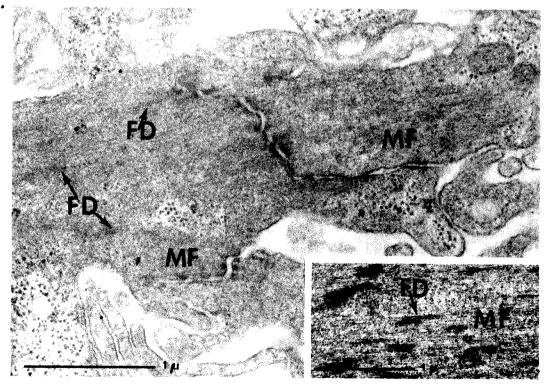


Fig. 6 (Machemer, van Horn, and Aaberg). Typical cell with dilated rough endoplasmic reticulum (RER), accumulations of glycogen (G), and marginally located intracellular filaments (F). Inset shows filaments at higher magnification.



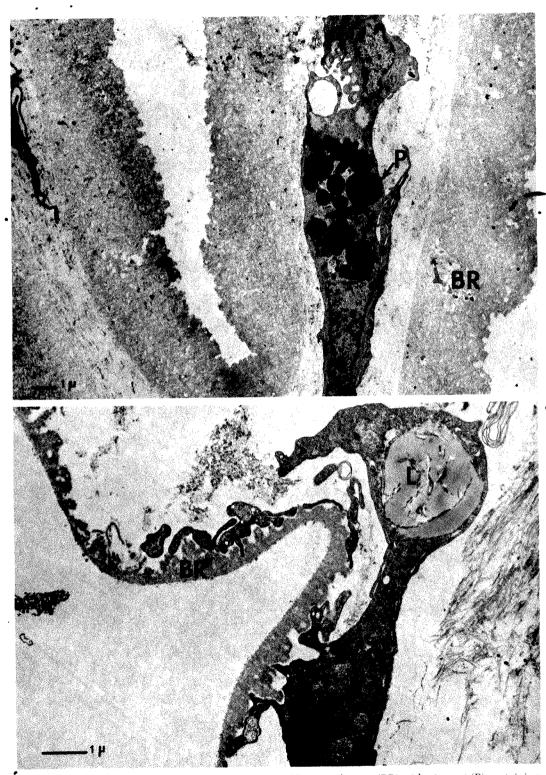


Fig. 8 (Machemer, van Horn, and Aaberg). Top, Basal lamina of retina (BR) with pigment (P) containing cell on vitreal surface of lamina. Note that some pigment granules are wedge-shaped. Bottom, Basal lamina of retina (BR) with adherent cell on retinal side. Large lipid-like droplet (L).

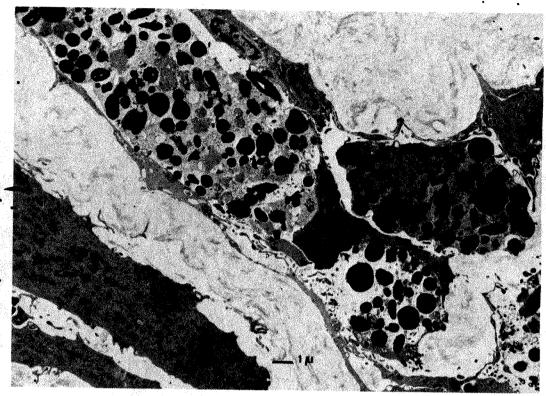


Fig. 9 (Machemer, van Horn, and Aaberg). Pigment-laden macrophages at various stages of vitality. Note normal and wedge-shaped pigment granules.

What then are the cells found in MPP? Many features suggested a cell of fibroblastic origin: the elongated shape of many cells, intracellular filaments including what appeared to be myofilaments, the abundance of dilated cisternae of rough endoplasmic reticulum indicating a metabolically active stage, and the finding of extracellular fibrils, presumably collagen.

However, other features such as microvilli, cell junctions, basement membrane formation, in other words, a polarization of the cell, and nonmembrane bound, often wedge-shaped pigment granules in the cytoplasm support the idea that the cells are of epithelial origin.

Previously, we investigated the proliferation of cells in experimentally induced retinal detachment in the owl monkey

eye. 1 There is a striking similarity between the findings in the experimental situation and in the human surgical specimens of the present study. Cells with the characteristics outlined above were dominant in the animal model as well (compare Figs. 3 and 4 with Fig. 10, top). To identify the origin of the cells, we had designed experiments in which pigment epithelial cells from one eye were autotransplanted into the vitreous cavity of the other eye. To avoid the possibility of contamination with cells of other origins, the cells were kept in cell-tight diffusion chambers.7 We came to the conclusion that pigment epithelial cells indeed were able to change their morphologic appearance. They either looked like macrophages (pigment epithelium macrophages), or were similar to fibrocytes (fibrocyte-like

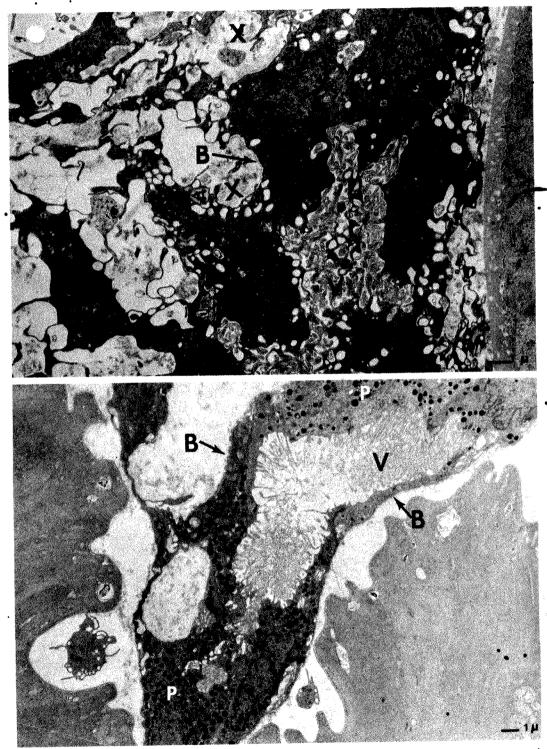


Fig. 10 (Machemer, van Horn, and Aaberg). Top, Preretinally proliferating cells in experimental detachment of the retina in owl monkey. Note similarity to Figures 3 and 4. Bottom, Tubule formation of preretinally located pigment epithelial cells in experimental retinal detachment. Compare with Figures 10, top, 3, and 4.

cells), or regained or retained pigment epithelium cell characteristics. They also produced extracellular filamentous material, presumably collagen.⁷

Just as in the experimental situation, some cells in the human MPP eyes appear as macrophages, many of which seemed to die. Their debris is phagocytosed by other macrophages, ever increasing the amount of pigment granules in these cells. Other cells have the appearance of Ebrocytes; most of the cells, however, rentind one of polarized pigment epithelium cells, which may appear in a rosette-like arrangement or even suggesting tubule formation (Figs. 4 and 10).

The masses of filamentous material in the vicinity of these cells presumably is collagen produced by these cells. Indeed, pigment epithelial cells are able to produce collagen. 7,8 Another argument for this interpretation is that collagen is not randomly distributed between cells. This would be true if the cells had grown into preexisting collagen-containing matrix. Instead, the filaments are found accumulating on the side of the cell where basal lamina is found, whereas the side where microvilli are located is electron empty. Also, the fibrils found in the present specimens are smaller (predominantly 12 nm in diameter) than the collagen fibrils normally found in human eyes (15 to 20 nm in diameter).9

Since the animal experiment and the human findings correspond so well, we believe many of the cells found in the human specimen are indeed of retinal pigment epithelial cell origin.

Other sources of cells, however, cannot be excluded. Glial cells might possibily have an appearance similar to the cells we have described as deriving from pigment epithelium. Glial cells are known to form basal lamina, have cell junctions, and exhibit polarity. However, we believe a glial membrane has a distinctly different morphologic appearance. In human

MPP,5 in experimental retinal detachment,10 and in spontaneous human preretinal proliferation,11 the cells of glial proliferations possess densely packed, long and coarse cell processes in parallel array with few nuclei, have rather few cell organelles, and they are rich in loosely packed cytoplasmic filaments and microtubules. Membranes formed by these cells can be polarized. Such cells at the viteous surface have junctions and microvilli and cells near the base of the membrane a discontinuous basal lamina look-. ing much like the junctions found in the outer limiting membrane of the retina. Glial membranes are confined to the surface of the retina and can often be traced back to breaks in the internal limiting membrane. 10.11 Constable and associates4.12 think all the fibroblastic membranes are of glial origin.

Proliferating vascular cells also demonstrate basement membranes and cell junctions. However, in animal experiments vascular proliferations beginning in the retina were never observed as a source of the MPP cells. There were also no vessels in the preretinal membranes of massive periretinal proliferation. Thus, it is unlikely that the cells derived from proliferating vascular cells.

Finally, there remains the possibility that some as yet unidentified cell type might also participate in the intraocular proliferative process. Smith, van Heuven, and Streeten¹³ found pigmented cells with obvious polarity in an eye operated on for proliferative retinopathy, a disease in which pigment epithelial cells presumably do not reach the vitreous cavity unless there is a retinal hole.

What of the importance of the abundantly appearing cytoplasmic filaments?² The exact function is presently not known. The large (10 nm in diameter) filaments are thought to participate in contraction of the cell as well as to have a cytoskeletal function.¹⁴ The thinner fila-

ments with fusiform densities are possibly myofibrils. The fixation technique applied for our specimens is inadequate to confirm this possibility. Fibroblasts in granulation tissue may contain extensive cytoplasmic filaments that show characteristics typical of smooth muscle. These cells are called myofibroblasts.15 The cells are usually found in young granulation tissue, but not in a complete scar. It has also been shown that similar myofibrils can occur in retinal pigment epithel- ium.¹⁶ Myofibrils are also thought to play an important role in the contraction of cells. Possibly the fibrils observed in our specimens could account for the contraction of the intraocular membranes found in MPP. The relative scarcity of the myofibrils may be an expression of the age of the intraocular scar tissue. One must remember that surgical treatment of MPP is done not in the early stages of this disease when myofibrils may be plentiful, but rather, after contraction has occurred and the scars of folded retina have stabilized.

SUMMARY

Biopsy specimens from vitreous and preretinal membranes, obtained during vitreous surgery from 39 human eyes suffering from massive periretinal proliferation, were examined electron-microscopically. Analysis of the cellular membranes demonstrated mostly cells with epithelial characteristics: polarization of the cells. basal lamina formation, specialized cellular junctions, and microvilli formation. These epithelioid cells contained prominent rough endoplasmic reticulum, glycogen deposits, a multitude of cytoplasmic filaments, some resembling myofilaments, and nonmembrane bound, sometimes wedge-shaped pigment granules. Macrophages were interspersed in the membranes. There was a striking similarity of these findings to those of an experimental model of retinal detachment in owl monkeys. We concluded that most likely the described cells derived from cells of pigment epithelial origin. .

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VISUAL FUNCTION IN ACUTE POSTERIOR MULTIFOCAL PLACOID PIGMENT EPITHELIOPATHY

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Acute posterior multifocal placoid pigment epitheliopathy (APMPPE) was first described by Gass in 19681 and has since attracted considerable attention. In this disease, acute onset occurs and the ocular fundus•shows flat, yellow-white lesions at the level of the retinal pigment epithelium at the posterior pole. The lesions spontaneously, but residual resolve changes in the pigment epithelium remain. If the lesions are near the macula, visual acuity is affected. Central and pericentral scotomas are present. Acute posterior multifocal placoid pigment epitheliopathy is a relatively benign disorder; most previously reported cases demonstrated moderate visual acuity loss and good recovery. 1-10 However, some patients had severe visual acuity loss and full recovery did not occur.3.6

Recently, a patient in the acute phase of APMPPE demonstrated an abnormal color match. This can arise solely by alteration in the width or spectral position of one or more of the absorbing spectra of the cone visual pigments¹¹ or by accumulation of a spectrally selective absorbing substance in the preretinal media.¹² We completed detailed clinical studies, measurement of dark adaptation, analysis of

color matching, and measurement of the Stiles-Crawford effect.

CASE REPORT

A 19-year-old woman reported here on • Feb. 3, 1976, one week after she noted blurred and decreased vision. Her visual acuity was R.E.: 6/7.5 (20/25), and L.E.: 6/12 (20/40). Ophthalmoscopic examination revealed circular white patches deep in the retina (Fig. 1, top left). At the fovea, both an acute lesion and a lesion that was pigmented and apparently in resolution could be noted. On fluorescein angiography, the early venous phase showed hyperfluorescence of the acute lesions and window lesions in the older area but no leakage (Fig. 1, middle and lower left). The lesions started to resolve almost immediately. Central vision returned to R.E.: 6/6 (20/20) and L.E. 6/7.5 (20/25) within two weeks. At three weeks, all the lesions had resolved; this left areas of pigmentation and depigmentation (Fig. 1, top right). Widespread defects of the pigment epithelium were evident on fluorescein angiography and the early phase showed areas of depigmentation and pigment proliferation (Fig. 1, middle right). The late fluorescein showed staining, but no leakage (Fig. 1, lower right). The fluorescein angiography was repeated at two months and seven months and showed no further gross change. There is still a widespread defect of the pigment epithe-

Visual fields—The visual fields measured on Feb. 4, 1976, showed large pericentral scotomas for a 1-mm white target (Fig. 2). During a one-year period, the visual fields gradually improved. We

From the Eye Research Laboratories, University of Chicago. This study was supported in part by the United States Public Health Service Research Grant EY 00901, Research Career Development Awards EY 70652, and EY 70663, and Project Grant EY 00523, from the National Eye Institute. Presented at the annual meeting of the Association for Research in Vision and Ophthalmology, Sarasota, Florida, April 26, 1977.

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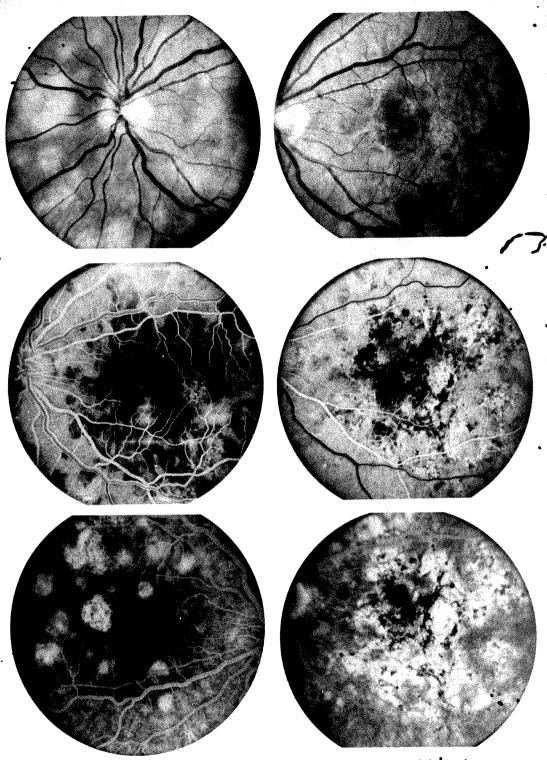


Fig. 1 (Smith and associates). Left side shows data during the acute stage. Top left, Fundus appearance showing round white placoid lesions at the posterior pole. Middle left, Fluorescein angiogram in the early venous phase with hyperfluorescence of acute lesions and window lesions in a resolving lesion. Bottom left, late fluorescein angiogram with staining of both acute and resolving lesions. Right side shows data three weeks after the acute stage. Top right, Fundus appearance showing areas of pigmentation and depigmentation. Middle right, Fluorescein angiogram in early venous phase shows widespread window defects and staining of the pigment epithelium. Bottom right, Late fluorescein angiogram with extensive staining of the pigment epithelium.

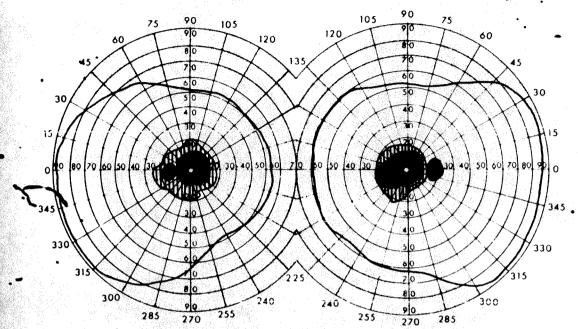


Fig. 2 (Smith and associates). Visual fields of the patient in the acute phase. (Feb. 4, 1976).

measured the central visual fields with a 2-mm white target (Fig. 3). By six months, the defect consisted of small parafoveal scotomas, with little further improvement after a year.

Retinal function evaluation—We completed clinical color vision testing, ¹³ electroretinogram (ERG), ¹³ electro-oculogram (EOG), ¹³ and dark adaptation testing on Feb. 4, 1976.

Clinical color testing included the Farnsworth Panel D-15 test and measurement of the Rayleigh equation with a Nagel anomaloscope. The patient made an abnormal color match. She needed more red in the mixture field than the color-normal observer; the matching range was wider than normal. She had an abnormal error score, above 600, on the Farnsworth Munsell FM-100 hue test without dominant axis.

The electroretinogram (ERG) showed a minimally subnormal b-wave; the EOG of R.E.: 1.53, and L.E.: 1.55 was subnormal (minimum normal value, 1.8). Dark adaptation was performed by using a 1-

degree target at 15 degrees to the superior retina. The time course of dark adaptation could not be measured in the right eye. This failure may have been caused by the test field falling within the area of pericentral scotoma. The left eye, tested after 45 minutes in the dark, showed a nearnormal final threshold.

The EOG was repeated three weeks later and found normal. Dark adaptation was measured in the right eye two weeks after the acute phase. There was a slow cone function, delayed rod-cone break at 22.5 minutes, and slow rod function (Fig. 4). Thresholds were approaching normal range (vertical bar on Fig. 4) by 45 minutes of testing and, after an hour in the dark, we measured good final thresholds. These abnormalities gradually improved during a one-year period. By seven months, cone function appeared normal, but rod adaptation was still delayed. The rod-cone break time approached normal limits by 13 months. The dark adaptation data indicate that the disease process affected the time course of the visual regen-

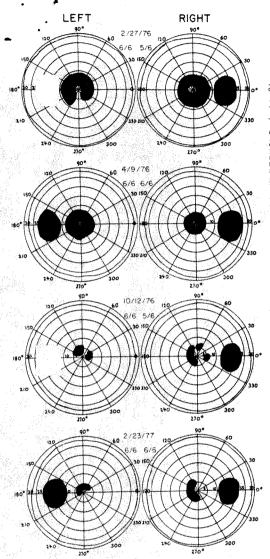


Fig. 3 (Smith and associates). Central visual fields measured with a 2-mm white target at successive intervals during recovery.

eration cycle, but the photopigments did regenerate. We made dark adaptation measurements at an area that was within the pericentral scotoma during the acute phase. One previous study noted normal dark adaptation on follow up,⁸ but it is not clear whether adaptation was measured in an affected area.

Color matching—For these experiments the Moreland Universal Anomalo-

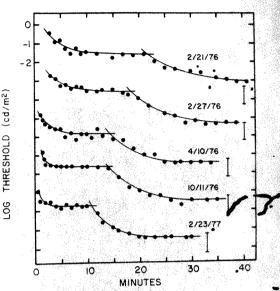


Fig. 4 (Smith and associates). Dark adaptation functions measured at successive intervals during recovery. A 1-degree target at 15 degrees to the superior retina was used. The figure shows the visual threshold in cd/m² plotted as a function of time in the dark. Successive functions are arbitrarily displaced for clarity. The vertical bar shows the normal interobserver range of the final dark-adapted threshold.

scope was used. Modifications of the instrument^{14,15} and its use as a clinical tool¹⁶ have been described. We investigated the color match area effect ¹⁴ and extended Rayleigh match series.¹⁶ Data are reported as the log green/red at the extremes of the matching range. We evaluated color matching performance at one week, three weeks, six weeks, two months, seven months, and 13 months after the initial visit.

The color matches are plotted as a function of field size for the 589-nm test field (Fig. 5). The interobserver range previously reported for ten observers aged 18 to 35 years is shown for comparison. Three days after the initial visit, the matches were displaced to red for all field sizes; they show only poorer discrimination with reduction in field size. The extended Rayleigh match series gave results similar to those of a bleached normal

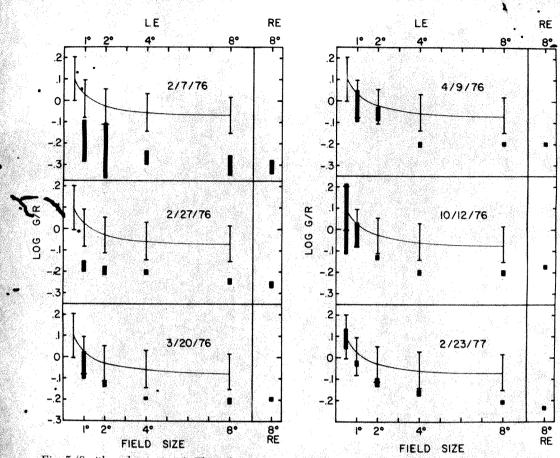


Fig. 5 (Smith and associates). The color-match area effect for the patient at successive intervals during recovery. The log green/red is plotted as a function of field size. The average function and interobserver range is shown for comparison.

eye.16 The small-field matches gradually recovered into the normal range, but the 4- and 8-degree matches remained displaced to red. These color match data are consistent with the field data showing persistent parafoveal defect, with virtually full recovery of small foveal field data. The shifted color match observed by us and in one previous study8 is similar to that made by a normal observer after a strong bleach of the visual photopigments.14. 17. 18 Bleaching the photopigments with a bright light reduces the effective optical density, thus narrowing the absorption spectra of the visual photopigments. Our results indicate that the

visual photopigments at the site of the lesion had reduced effective optical density.

Stiles Crawford—The experimental apparatus and calibrations ¹⁹ and our clinical procedures ¹⁶ have been described. The apparatus allows measurements of the Stiles-Crawford effect with a 620-nm, monochromatic center-annulus brightness matching procedure. The annulus, subtending 3 degrees and 38 minutes with field luminance of 130 cd/m², is centered on the optic axis of the patient's cornea. The central circle, subtending 55 minutes, is provided by a movable beam which can be shifted horizontally in the

plane of the patient's pupil. A telescopic viewing device²⁰ is used to align and monitor the position of the patient's eye throughout the experiment. Data are reported as log-relative efficiency of the central movable beam for various entry positions. The Stiles-Crawford effect was measured at one month, two months, seven months, and 13 months after the initial visit.

The first test revealed little change in brightness match as we changed the · pupil-entry position, until the very edge of the pupil (Fig. 6). The central 6 mm of a normal Stiles-Crawford function can be adequately described by a parabola.21 More eccentric entry leads to a flattening of the function20; this can be observed in persons whose Stiles-Crawford effect has an eccentric peak. 22.23 A severe disruption of the receptoral layer means the majority of light rays enter diagonally for all pupil-entry positions, thus giving a flat Stiles-Crawford effect. Our data confirm that a severe disorientation of photoreceptors existed. The Stiles-Crawford effect gradually recovered in subsequent months. By seven months, we measured a Stiles-Crawford effect function centered near the optic axis. This function was somewhat wider than the normal Stiles-Crawford effect. At 13 months, the Stiles-Crawford effect function was well centered and looked normal.

DISCUSSION

Our studies revealed three abnormalities associated with the lesion that have not been previously emphasized for APMPPE: (1) an abnormality of the time course of dark adaptation indicated a metabolic disfunction of the retina; (2) an abnormal color match reflected a reduction in the effective optical density of the cone visual photopigments; and (3) an abnormal Stiles-Crawford effect indicated a profound disorientation of the visual photoreceptors.

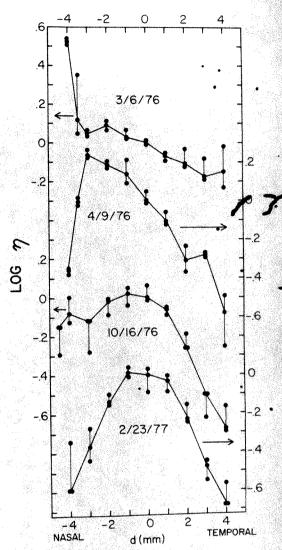


Fig. 6 (Smith and associates). The Stiles-Crawford effect measured on the patient's left eye at successive intervals during recovery. The log-relative luminous efficiency is plotted as a function of the pupil-entry position. Arrows point to the appropriate ordinate scale for each function.

The clinical description and defects of retinal function are typical of these in previous reports. 1-10 Our observer has shown full recovery of the central 2 degrees of foveal vision, but has persistent parafoveal field defects. 1,3,6,10 The functional defects that showed recovery did so on a parallel time course that lasted over one year and was much slower than the

inflammatory process. Fluorescein angiography showed residual widespread and unchanging abnormality of the pigment epithelium. These results are also typical of those in previous reports. Thus, although we studied only one observer, we believe our findings serve as a model for the defects and recovery of visual function in APMPPE.

Similar abnormalities of the Stiles-Crawford effect and color match have been described for central serous choroidopathy and could be explained by mechanical disturbance of the photoreceptors.16 The one-year recovery period observed in our patient is much longer than recovery after laser treatment for serous elevation. This fact, together with the different time courses for the resolution of the inflammatory process and the recovery process for visual function, suggests that visual function loss in APMPPE is more than a purely mechanical defect. Previous reports have been divided as to whether APMPPE is primarily an epitheliopathy 1,3,8 or primarily a choroiditis. 2.4-7.9-10 Our data indicate a primary and longlasting involvement of the visual photoreceptors and pigment epithelium. The inflammatory episode may be of choroidal origin. The recovery process we have described reflects the recovery of the epithelial and photoreceptor cells to their correct orientation and association with the processes of the pigment epithelium.24-26

SUMMARY

We examined a 19-year-old woman with acute posterior multifocal placoid pigment epitheliopathy one week after she noted blurring of central vision. Her corrected visual acuity was R.E.: 6/7.5 (20/25), and L.E.: 6/12 (20/40). The visual fields showed 10-degree pericentral scotomas. A color vision defect and an abnormal Stiles-Crawford effect were present. Dark adaptation showed a delayed time

course, with normal final thresholds. The electro-oculogram was subnormal; suggesting widespread abnormality of the retinal pigment epithelium.

Active lesions resolved within three weeks, but fluorescein angiography showed characteristic widespread residual changes. Within three weeks, she had normal visual acuity and a normal electro-oculogram. Other tests of visual function showed recovery with a slower time course. By one year, the visual fields, color matching, Stiles-Crawford effect, and dark adaptation were almost normal.

Analysis of the color-matching data and Stiles-Crawford effect indicated that the abnormalities of macular function were caused by an underlying lesion of the pigment epithelium and photoreceptors. This caused a physical distortion of the photoreceptor layer and metabolic disfunction of the photoreceptors.

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A SPECIFIC ENZYME DEFECT IN GYRATE ATROPHY

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Although the association of hyperornithinemia with gyrate atrophy of the choroid and retina has been reported in about 30 patients, 1-4 evidence for the presence of an enzyme defect has not been previously reported. We report herein the marked deficiency of ornithine aminotransferase in transformed lymphocytes of a patient with gyrate atrophy, and intermediate values in her daughter, an obligate heterozygote, as compared with normal values.

CASE REPORT

Case 1—A 34-year-old white woman was first seen at the National Eye Institute in June 1977 because she had retinal degeneration of long duration. The patient had had three siblings: One sister died at 2 years of age of pleurisy and had no known visual problems. One brother who committed suicide at 29 years of age had had a history of poor vision since childhood characterized by night blindness and deterioration of vision with age. A 37-year-old sister is normal. The patient has a 3-year-old daughter who is considered normal. There was no history of any other family members with ocular disease. The family is of Portuguese descent and consanguinity was denied.

The patient stated she had had poor eyesight since about 7 years of age when glasses were prescribed

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for myopia. A progressive decrease in vision accompanied by poor peripheral vision was noted. In 1968, at 25 years of age, a diagnosis was made of retinal degeneration with bilateral cataracts. She underwent uncomplicated cataract extraction in both eyes. In 1973, an intraocular hemorrhage occurred in the right eye with gradual clearing but with residual poor visual acuity of counting fingers. The vision in the left eye has remained stable. She had no medical complaints and her general health was good.

The positive physical findings were related to the ocular examination. The best corrected visual acuity was R.E.: counting fingers, with a refractive correction of +9.50 +0.50 × 180 degrees, and L.E.: 6/9 (20/30) with a +9.25 sphere. External examination indicated right exotropia of 15 prism diopters. Slitlamp examination revealed deep anterior chambers bilaterally and surgical aphakia with iridodonesis. Intraocular pressure on applanation tonometry was 15 mm Hg in each eye. Ophthalmoscopic examination of the right eye revealed a hazy media, and an oval optic disk with waxy pallor and a drusen at the 5:00 o'clock position (Fig. 1). A clump of pigment was noted in the macular area; and, in the midperiphery, were confluent areas of chorioretinal atrophy with scalloped margins. In the left eye, the disk had a waxy pallor and a heavy clump of pigment was adjacent to the macula. The midperipheral areas of chorioretinal atrophy were identical to those in the right eye (Fig. 2).

Goldmann visual fields in the right eye showed a 20-degree field with a 10-degree central scotoma using the V₄e test object (Fig. 3). In the left eye, there was a 20 to 30-degree constricted field using V₄e test object and a paracentral scotoma below fixation which spared the fovea (Fig. 3). Psychophysical testing was performed. With the right eye, she failed the HRR plates, Panel D-15, and the Nagel anomaloscope test, but identified bright Holmgren wools. With the left eye, she failed the HRR plates and Nagel anomaloscope, arranged the Panel D-15 slowly with small errors, and named and matched the Holmgren wools. Dark adaptation proceeded slowly with best rod thresholds in either eye being elevated about 2.3 log units. Cone thresholds could not be

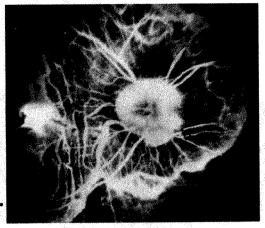


Fig. 1 (Kaiser-Kupfer, Valle, and Del Valle). Fluorescein angiogram demonstrating drusen of the right optic nerve at arrow.

measured. The electroretinogram showed no measurable rod or cone responses and the electrooculogram was abnormal with no measurable difference between the light and dark.

Fluorescein angiography demonstrated an absence of the choriocapillaris flush phase in those scalloped areas of pigment epithelial thinning and loss, while adjacent areas of more normal appearing pigment epithelium about the optic nerve showed a more normal choriocapillaris flush phase (Fig. 4).

Case 2—We examined the patient's 3-year-old daughter; she had no physical or ophthalmologic abnormalities.

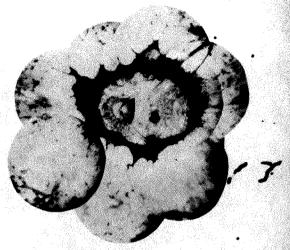


Fig. 2 (Kaiser-Kupfer, Valle, and Del Valle). Left eye. Montage of posterior pole and periphery.

METHODS

Peripheral blood lymphocytes were prepared from fresh heparinized whole blood obtained from our patient (Case 1), her daughter (Case 2), and three healthy control subjects (two men and one woman) whose ages ranged from 22 to 33 years. Lymphocyte tranformation studies

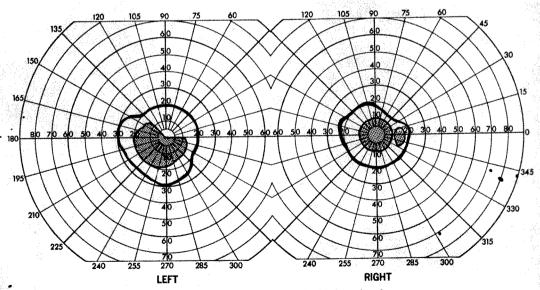


Fig. 3 (Kaiser-Kupfer, Valle, and Del Valle). Visual field of left and right eyes.

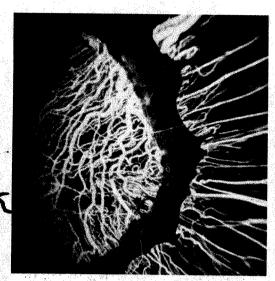


Fig. 4 (Kaiser-Kupfer, Valle, and Del Valle). Fluorescein angiogram of left eye. Normal filling of choriocapillaris is present in area near the optic nerve, but absent in more peripheral regions of pigment epithelial thinning and loss.

and thymidine incorporation with and without added phytohemagglutinin were performed as described previously.⁵

Ornithine aminotransferase activity⁶ and Δ¹-pyrroline-5-carboxylic acid dehydrogenase⁷ were assayed by previously described radioisotopic methods. Ornithine decarboxylase activity of lymphocyte extracts was assayed by measuring the ¹⁴C-CO² liberated from ¹⁴C-C¹-ornithine as described by Nissley, Passamani, and Short.⁸ Protein was measured by the method of Lowry and associates.⁹

RESULTS

The mean fasting plasma ornithine concentration in one patient (Case 1) was $964+79~\mu m/l$, approximately 15-fold higher than normal adult values. High voltage electrophoresis of her urine revealed a marked increase in the ornithine-lysine spot and a minimal increase in cystine. These biochemical abnormalities together with the typical ophthalmologic findings confirmed the

diagnosis of gyrate atrophy. Her daughter, Case 2, had a normal plasma ornithine concentration of 56 µm/l, as expected for an obligate heterozygote. With regard to lymphocyte transformation studies. 3Hthymidine incorporation was measured to determine if all the lymphocyte samples underwent transformation. There was no difference among the samples from the patients or the normal controls in the response to phytohemagglutinin. The thymidine incorporation of cultures without phytohemagglutinin was 863±509 • cpm/culture (mean of all samples) as compared to 183.53±10.902 cpm/culture in those grown with phytohemagglutinin. Thus, there was a 200-fold increase in ³H-thymidine incorporation; by this criterion all lymphocyte samples (Cases 1 and 2 and controls) were successfully transformed.

A comparison of the ornithine aminotransferase activity in lymphocytes of patients (Cases 1 and 2) and controls, however, revealed striking differences (Fig. 5). The specific activity of ornithine aminotransferase in control lymphocytes in-

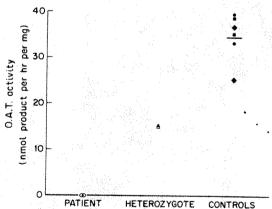


Fig. 5 (Kaiser-Kupfer, Valle, and Del Valle). Ornithine aminotransferase activity in transformed lymphocytes. The lymphocytes from each individual were assayed in triplicate on two separate occasions. Each point represents the mean of a triplicate determination; the horizontal bar denotes the mean of the control values.

creased dramatically with transformation; the mean activity was 34.6 ± 5.1 nm of Δ^1 -pyrroline-5-carboxylic acid produced per hour per milligram of protein. By contrast, the lymphocytes of our patient, Case 1, had no detectable ornithine aminotransferase activity. The ornithine aminotransferase activity in the lymphocytes of the daughter, Case 2, was 15.1 ± 2.2 nm of Δ^1 -pyrroline-5-carboxylic acid produced per milligram of protein or 44% of the mean control value.

All attempts to demonstrate some residual ornithine aminotransferase activity in the lymphocytes of the patient (Case 1) by altering the assay conditions were unsuccessful. Furthermore, no evidence was found for an ornithine aminotransferase inhibitor in the cells of this patient. To confirm further the specificity of the ornithine aminotransferase deficiency in gyrate atrophy, an assay was performed for Δ1-pyrroline-5-carboxylic acid dehydrogenase, an enzyme located primarily in the mitochondrial matrix¹⁰ along with ornithine aminotransferase. There was no difference between Δ^1 -pyrroline-5-carboxylic acid dehydrogenase activities between control lymphocytes and the lymphocytes from our patient (Case 1).

Additionally, we measured ornithine decarboxylase activity because a deficiency of this enzyme might be expected to result in hyperornithinemia. The activity of ornithine decarboxylase in control lymphocytes was indistinguishable from that of the lymphocytes of the patient in Case 1.

DISCUSSION

Takki and Simell² and McCulloch and Marliss¹¹ suggested that a deficiency of ornithine aminotransferase may be responsible for the hyperornithinemia present in gyrate atrophy. This mitochondrial matrix enzyme catalyzes the pyridoxal phosphate-dependent transamination of ornithine to Δ^1 -pyrroline-5-carboxylic

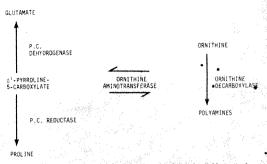


Fig. 6 (Kaiser-Kupfer, Valle, and Del Valle). Schematic diagram of ornithine metabolism; P. C. designates Δ¹-pyrroline-5 -carboxylic acid.

acid (Fig. 6). However, no measurement of ornithine aminotransferase activity in tissues or cells of patients with gyrate atrophy have heretofore been published.

Previous studies have shown that low levels of ornithine aminotransferase activity are normally present in circulating lymphocytes. During lymphocyte transformation, there is a 15-fold increase in the specific activity of ornithine aminotransferase to levels that can be easily and reliably measured. Utilizing these conditions, the activity of ornithine aminotransferase was measured in lymphocytes of a patient with gyrate atrophy. We found a marked deficiency of ornithine aminotransferase in transformed lymphocytes of the patient. The patient's daughter, who had no ocular signs or symptoms of gyrate atrophy, demonstrated a 56% reduction in ornithine aminotransferase activity in her lymphocytes. Because the daughter is an obligate heterozygote in this autosomal recessive disorder, her intermediate value in the level of ornithine aminotransferase activity is further evidence that a deficiency of ornithine aminotransferase is the primary defect in gyrate atrophy. Additionally, normal activity of Δ^1 -pyrroline-5-carboxylic acid dehydrogenase, which, along with ornithine aminotransferase, is primarily in the mitochondrial matrix, indicates the reduction in ornithine aminotransferase activity in gyrate atrophy is not secondary to

some structural abnormality of the mitochondria.

A block in ornithine metabolism at the ornithine aminotransferase reaction explains the hyperornithinemia associated with gyrate atrophy (Fig. 6). Although the ornithine aminotransferase reaction is reversible, the equilibrium constant is 70fold in favor of the formation of Δ^{1} pyrroline-5-carboxylic acid. 12 A block in this reaction world result in ornithine accumulation.

The clinical appearance of gyrate atrophy has been known for about 80 years.13 Four years ago, Simell and Takki1 established the presence of large increases in plasma ornithine levels with gyrate atrophy. The present study has now identified an absence of ornithine aminotransferase as the specific enzyme abnormality associated with gyrate atrophy.

SUMMARY

To establish the enzyme defect in gyrate atrophy, we measured the activity of ornithine aminotransferase in phytohemagglutinin stimulated lymphocytes in a patient with gyrate atrophy, her daughter. and three normal controls. The patient's cells had no detectable ornithine aminotransferase activity and the daughter's cells had 44% of control activity. This intermediate value is characteristic of an obligate heterozygote. These results are the first demonstration of an enzyme defect in gyrate atrophy.

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AN IMPROVED MICROSURGICAL CEILING-MOUNTED UNIT AND AUTOMATED TELEVISION

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The increasing complexity of microsurgical ophthalmic microscopes led to the development of ceiling-mounted microscopes¹⁻³ to avoid space-taking floor-mounted columns. The problem of accessory instruments leading to a mass of cables on the floor and multiple wheel carts around the surgeon still remains unresolved.

However, once a ceiling-mounted structure is installed, it can hold various additional instruments. This potential was realized from the beginning by Barraquer. But, Draeger³ was the first to design a multipurpose combination with built-in suction pump for phacoerysis, cautery, diathermy unit, and microphone. Existing ceiling-mounted combination microsurgical units have no versatility for modification or improvement. Therefore, we asked the American Sterilizer Company to redesign their existing ceilingmounted microsurgical column (Fig. 1), which was originally based on a model developed at the Jules Stein Institute.2

We report herein our experience with five of these units after one year of use. The following list comprises our requirements for the redesign:

Capability to build in a variety of additional instruments of the surgeon's

choice—This problem has been solved by providing a large instrument box (Fig. 1, a) with removable panels into which various commercially available instruments can be installed. This instrument box is above the level of the microscope arms. It can be lowered for easy access to the instruments.

For our purposes we selected a cryosurgical unit, a wet-field bipolar coagulator, and a high-intensity fiber optic twin light source. All supply connections to these instruments run through the column and most units are activated by footswitches.

Having various instruments built into the microsurgical unit has worked well. The instruments are always quickly available. Annoving cables and connections are avoided. The instruments selected should be built-in in their commercially available form for easier repair and exchange. We mistakenly separated parts of the cryosurgical unit, which made repair difficult. Long supply lines for the cooling gas to the cryosurgical unit necessitated a pressure regulator in the column. Pressure regulation is usually done at the supply tank. In our situation the gas supply is outside the operating room. The instrument box is made so that instruments can be exchanged for improved versions.

Mirror image design—It is important that preferences about which side instruments, switches, knobs, and plugs should be on for a given operating room can be followed. One may elect to have all the instruments handled by the circulating nurse on one side and those handled by

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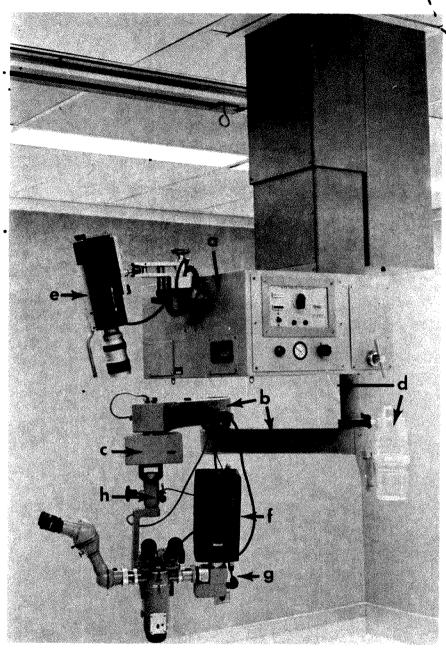


Fig. 1 (Machemer and Parel). Microsurgical ceiling-mounted combination unit. a, Large instrument box in lowered position with built-in cryosurgical unit, wet-field coagulator visible. On the other side is a fiber optic light source and a radiofrequency diathermy unit. b, Horizontal support arms with panels for plugs and switches. c, X-Y translation stage with operating microscope attached to it. d, Vacuum line with suction bottle. e, Survey television camera with short, horizontally adjustable support arm. f, Microscope television camera. g, Automatic diaphragm control. h, Microphone.

the scrub nurse on the other side. Except for the tightening screws on the microscope arms, this principle has been accomplished in our modified units and has worked out well.

Two sturdy horizontal arms for the microscope attachment with multiple outlets—To reduce potential vibration, the horizontal arms that carry the operating microscope should be sturdy, have as few joints as possible, and be as short as possible (Fig. 1, b).

In the present design, the combined length of the arms is 91.5 cm (36 inches). Experience has shown that these arms are stable. Vibration is dampened in four to six seconds. The various switches and receptacles on the arms are easily accessible. Since the arms are hollow, all cables run through the arms into the microsurgical unit. Therefore, only short connections to the attached microscope and its many accessories are achieved. We selected additional connections for a microphone and for a photographic strobe light. If desired, further modifications can be made.

Capability to attach a motorized X-Y translation stage to the horizontal arms—The X-Y stage (Fig. 1, c) allows electrical horizontal adjustment of the microscope and is controlled by a footswitch. It avoids the need for manual microscope adjustments and thus makes the operating microscope completely automatic.⁴ Therefore, the operating microscope does not need a sterile cover during operations. Also, a specialized operating table that permits horizontal movements of the patients, as proposed by Draeger,⁵ is no longer necessary.

Vacuum for aspiration from surgical field—A vacuum outlet with a regulator is built into the body of the unit. The collector bottle (Fig. 1, d) is attached to the base of the column by a quick release system. This feature has eliminated the need for an across-the-room suction tube. For safe-

ty reasons, it is totally separate from the vacuum used for anesthesia.

Additional 110-V AC electrical outlet—These outlets at the back of the column provide for any unforeseen electrical appliance and, thus, prevent the necessity of running cables across the operating room. Unfortunately, the Florida electrical safety code requires special twist-lock Hubbel plugs and sockets which make the connection difficult.

Floor-mounted outlets and footpedals—The various functions of the operating microscope, the cryosurgical unit, the wet-field bipolar coagulation unit, and the operating room light switches are operated with footswitches. To eliminate a multitude of cables connecting the microsurgical unit with these activating footpedals, all cables enter the room from under the floor and terminate in a floormounted low voltage outlet. Space for future variations with additional outlets is provided (Fig. 2, a).

Initially, we left the various pedals separated under the operating table. But, we soon lost total control of the various positions, which caused major confusion. Also, damage during cleaning of the operating room was unavoidable. We solved these problems by having all footpedals bonded to a common metallic frame (Fig. 2, b). However, the weight of this combined footswitch has caused some problems in manueverability of the footswitches, especially when used in nonmicrosurgical procedures.

Provision for outlets and attachments of two television cameras and provision for microphone connections—Televising ocular surgery requires a survey view of the operative field, as well as an enlarged view through the operating microscope. This necessitates a camera equipped with an objective as long as survey is desired. Later, the camera is attached to the operating microscope after removing the objective when microsurgery is recorded.

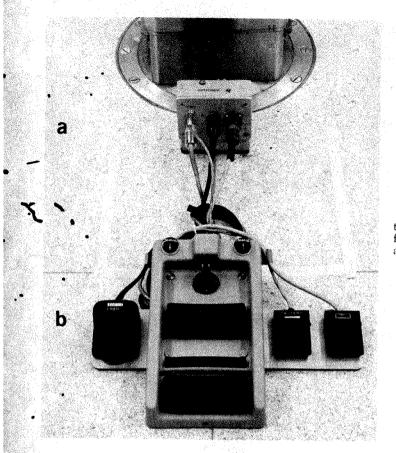


Fig. 2 (Machiner and Parel). At the base of the operating table is a floor-mounted multiple outlet (a) and combined footswitch (b).

This constant change is not practical during an operation. It requires a person to handle the camera and causes damage to the equipment.

We therefore designed a system of two cameras that are pre-aligned. The first camera, attached to the instrument box and used for survey view, is prefocused at the beginning of the operation once the instrument box is in low position (Fig. 1. e). The second camera is attached to the operating microscope by a beam splitter4 (Fig. 1, f). After use of the overhead camera, a simple switch in the television circuit is activated and switches over to the microscope camera. The microscope camera is automatically aligned and its image intensity automatically adjusts to the correct level, by a specially designed system (automatic diaphragm control)

(Fig. 1, g), when the surgeon focuses the microscope on the operating field. For control of the recording during surgery, a television monitor and videotape recorder are placed elsewhere in the operating room. A tiny microphone (Fig. 1, h) plugged into the horizontal arm and connected with the monitor provides the sound during videotape recordings.

This system has worked well for recording surgery, teaching, demonstrating to visitors, and especially in improving participation of the operating room crew in microsurgery.

The only known mistake made was that the overhead camera supporting arm was too low and too long. The objective of the camera hit the horizontal microscope arms and the camera sometimes recorded the back of the surgeon's head instead of the operative field. This was improved by elevating and shortening the attachment for the support arm of the camera.

Retractability of the microsurgical column—If an operating microscope is not needed, it should be fully retractable. However, the various built-in instruments should remain accessible. In our modified unit the microscope can be partially retracted while the instrument box was still in the lower position.

One unanticipated problem with using a multipurpose microsurgical column was that illumination of the operative field in retinal and plastic surgery became difficult, despite the use of ceilingmounted surgical lights on each side of the column. The wide body of the column blocked illumination from the foot of the patient. By placing the surgical lights above the lowered instrument box, and by using headlights, we have solved the problem in part.

SUMMARY

To increase the versatility of a ceilingmounted microsurgical unit, we instructed an instrument company to redesign an existant unit according to our requirements. The modified instrument has improved microscope supporting arms capable of supporting an inserted X-Y translation stage. The unit houses diathermy, cautery, fiberoptic light source, and suction devices. Most of these devices can be exchanged for others to adapt to individual requirements. Mirror image design increases versatility. mounted outlets avoid a multitude of cables. A double color television system for automatic recording of surgery has been installed.

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SYMBLEPHARON IN SARCOIDOSIS

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Cicatricial involvement of the conjunctiva including symblepharon is rare. The purpose of this case presentation is to document the association of symblepharon with systematic sarcoidosis and, thus, add ocular sarcoidosis to the causes of cicatricial conjunctivitis.

CASE REPORT

A 38-year-old black man complained of shortness of breath and ephiphora of four months' duration. Five years previously a diagnosis of sarcoidosis had been confirmed by biopsy of a scrotal mass and nasal polyps. He complained then of nasal congestion, painless swelling of the parotid glands, and a scrotal mass. An opthalmological evaluation had shown follicles involving the superior and inferior palpebral conjunctiva. The lacrimal glands were enlarged, visible, and associated with mild keratitis sicca. There was no uveal or retinal involvement. Biopsies of the left lacrimal gland and left inferior palpebral conjunctiva showed sarcoidosis. The patient was treated with artificial tears. The lacrimal gland biopsy did not appear to affect the progression of the keratitis sicca in the left eye.

The patient developed skin lesions of the face, arms, and legs over the next five years. Biopsy of the lesions had shown sarcoid, and they were treated with intralesional injections of triamcinolone acetonide with good results.

When I saw the patient, active skin lesions were confined to his arms and legs, and consisted of purplish papules which coalesced into well-demarcated, geographic areas. The lesions on the face (Fig. 1) appeared flat and were hyperpigmented from treatment with intralesional injections of triamcinolone acetonide. The eyelids were thickened, and a cicatrical entropion with trichiasis of the lower eyelids was evident (Fig. 2). There was no evidence of cranial nerve abnormality.

The best corrected visual acuity was R.E.: 6/12 (20/40), and L.E.: 6/6 (20/20). The conjunctiva revealed follicles and cicatrization of both upper and lower palpebral conjunctivae of both eyes (Fig. 3).

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Fig. 1 (Flach). Facial lesion after local corticosteroid injections.

Symblepharon was noted bilaterally, but was most prominent on the right-eye (Fig. 4). The lacrimal glands were not visible.

Examination of the cornea revealed a linear, blotchy epithelial keratitis involving the exposed area of the inferior cornea. An early arcus senilis was noted. There was no evidence of uveal or retinal involvement.

Results of laboratory studies revealed mild eosinophilia, a mildly elevated sedimentation rate, and a slightly elevated serum protein. The patient showed anergy to tuberculin of both intermediate and full strength, but the skin test was reactive for dermatophytin. Chest roentgenograms revealed a hilar adenopathy without evidence of peripheral fibrosis, although results of pulmonary function studies were consistent with obstructive lung disease. The patient had a weakly positive rheumatoid factor, and a slightly elevated glucose tolerance curve. Results of the following laboratory tests were unremarkable: serologic tests for syphilis, lupus erythematosus cell preparation, antinuclear antibodies, serum creatinine, and bilirubin levels. An electrocardiogram showed sinus tachycardia, occasional premature ventricular contractions, and low QRS voltage. X-ray films of the upper gastrointestinal tract revealed a gastric ulcer. Giemsa-stained conjunctival scrapings showed few eosinophils and rare Leber cells, but no evidence of epithelial inclusions. Repeat conjunctival scrapings on two subsequent occasions failed to show eosinophils, but demonstrated a few polymorphonuclear leukocytes, and Staphulococcus aureus was cultured from the specimen.

The patient was treated with artificial tears, soft contact lenses and excision of the symblepharon (which revealed only fibrosis); he then showed marked improvement in signs and symptoms (Fig. 5). The initial complaint of epiphora, which seemed



Fig. 2 (Flach). Right eye. Cicatricial entropion with trichiasis.

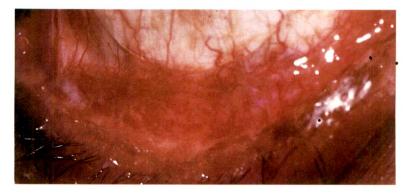
• Fig. 3 (Flach). Left eye. Cicatrization of upper palpebral conjunctiva.





Fig. 4 (Flach). Right eye. Lower eyelid symblepharon.

Fig. 5 (Flach). Right eye. Partially excised lower eyelid symblepharon.



related to the associated mucus deficiency, disappeared. Glucocorticoids were administered systemically, which alleviated the patients shortness of breath. Antacids were given for the gastric ulcer. A repeat roentgenogram showed healing.

Discussion

As early as 1931 Blegvad¹ described cicatrizing conjunctivitis caused by sarcoidosis. Crick's ² review of the ocular manifestations in sarcoidosis showed a photograph suggestive of cicatrization, but the caption described a mass of confluent follicles.

In the present case I found no evidence of a previous membranous, or pseudomembranous conjunctivitis, or chemical injury.3 The left inferior palpebral confunctival biopsy may have contributed to the conjunctival scarring on the left lower evelid, but there was marked cicatricial involvement of all four eyelids. Furthermore, the greatest symblepharon formation was present on the right eve. The cicatrization was not suggestive of that seen in chlamydial infestations, nor could I find evidence of *Chlamydia*. There was no evidence of collagen disease such as scleroderma, systemic lupus erythematosis, or polyarteritis nodosa. The patient had no signs of related dermatologic problems such as dermatitis herpetiformis, epidermolysis bullosa, erythoderma ichthyosiforme, acne rosacea, or exfoliative dermatitis.

The patient's age, the course of the conjunctival involvement, and the absence of dermatologic and oral involvement, other than sarcoid lesions, are unlike benign mucous membrane pemphigoid. Mucus membrane pemphigoid is considered an antibody-dependent disease on the basis of constant, local eosinophilia. In this case the single demonstration of conjunctival eosinophils, unconfirmed by repeat scrapings, suggests only intermittent eosinophilia, unlike mucus membrane pemphigoid.

The eosinophil is not specific for benign mucus membrane pemphigoid, but is characteristic of allergic inflammation and has been reported in association with *Staphylococcus aureus*. Although this patient lacked signs of staphylococcic blepharoconjunctivitis, *S. aureus* was cultured from his eyelids and conjunctiva, and he may be in a carrier state.

Results of all laboratory studies, including cultures and stains of all biopsy specimens and multiple sputum samples, revealed no evidence of tuberculosis.

Ocular involvement occurs in 63% of histologically proven cases of sarcoidosis. Keratoconjunctivitis sicca is frequent. This patient initially had Mikulicz's syndrome (enlargement of the lacrimal gland and parotid gland) and early keratoconjunctivitis sicca.

Although keratoconjunctivitis sicca and uveitis are the most frequent ocular findings in sarcoidosis, conjunctival involvement occurs in 2% to 25%.^{2,3} Sarcoid nodules simulating follicle formation in the conjunctiva are well documented, and biopsy of the involved area is a simple, relatively safe means of obtaining tissue for histologic diagnosis.¹³

SUMMARY

A 38-year-old black man had developed sarcoidosis, confirmed by biopsy five years earlier. He then developed skin lesions and, at age 38, follicles and cicatrization of the upper and lower palpebral conjunctivae of both eyes. This patient had keratoconjunctivitis sicca, lacrimal gland enlargement, and cicatrization of the conjunctiva with symblephardn. Biopsies of the lacrimal gland, conjunctiva, skin, nasal polyps, and epididymis all showed sarcoidosis.

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OPHTHALMIC MINIATURE

In Sushruta's classic Hindu treatise, the operation for cataract is given, including the advise to pierce the right eve with the left hand, and vice versa. If my tentative dates are right, this was an Indian, not an Alexandrian discovery.

> Guido Majno, The Healing Hand Cambridge, Mass., Harvard University Press, 1975

PROGRESSIVE NATURE OF PIGMENTED PARAVENOUS RETINOCHOROIDAL ATROPHY

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Two years ago, we reported three cases of pigmented paravenous retinochoroidal atrophy.¹ The progressive nature of the disorder was alluded to, but no direct documentation was available. Progression was inferred, because of increasing clinical involvement with age. Our youngest patient (Case 2) showed the least retinopathy, while the oldest patient

(Case 3) showed the most. Our first patient (Case 1) has since returned with dramatic changes in the posterior pole of both eyes. We think this is the first documented record of the striking functional and ophthalmoscopic deterioration that occurs in pigmented paravenous retinochoroidal atrophy with time.

CASE REPORT

A 27-year-old man, first seen in March 18/4, complained chiefly of diminished peripheral vision in each eye. In February 1977, his best corrected visual acuity was R.E.: 6/9 (20/30)-2, and L.E.:6/12 (20/40)+2. Goldmann fields were repeated (Fig. 1). They showed further loss of inner isopters: I₂ and I₃ isopters were absent. I₄ had decreased from 35 degrees (maximum radius) to 5 degrees on the left and from 10 to 5 degrees on the right. Dark adaptometry was not repeated.

The most impressive changes were noted ophthalmoscopically. Retinal pigment epitheliopathy was

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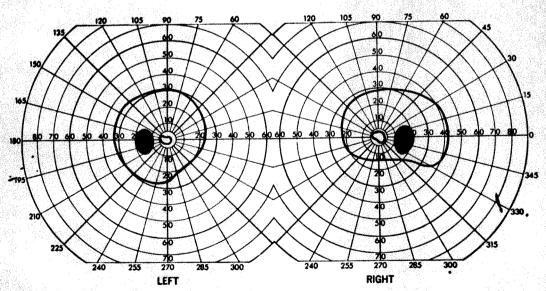


Fig. 1 (Pearlman, Heckenlively, and Bastek). Goldmann fields showing loss of inner isopters in both eyes, as compared to two years ago. Remaining isopters are IV₄ (the larger) and I₄ (the smaller).

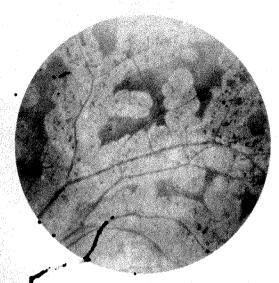


Fig. 2 (Pearlman, Heckenlively, and Bastek). Fundus showing "islands" of normal retinal tissue surrounded by confluent areas of increased retinochoroidal involvement. The scalloped appearance of the lesions resembles gyrate atrophy.

present in large areas that were previously normal. Abnormal-appearing lesions, once confined to a strict paravenous distribution, showed confluence with "islands" of normal retina occurring in relative isolation. Some areas had a distinctly scalloped appearance (Fig. 2), resembling a posteriorly situat-



Fig. 3 (Pearlman, Heckenlively, and Bastek). Localized atrophic areas with clear crystal deposition are depicted. Clumps of pigment deposition are noted in the fundus periphery.

ed gyrate atrophy of the choroid. Several localized atrophic areas with clear crystal deposition were noted (Fig. 3). Peripheral areas showed the greatest degree of pathological confluence, with moderate pigmentary deposition. No convincing optic nerve pallor or frank optic atrophy was evident. Other areas of the fundus still showed the characteristic paravenous nature of the basic disease.

DISCUSSION

The resemblance of clinically advanced pigmented paravenous retinochoroidal atrophy to gyrate atrophy, another rarely encountered (recessive) chorioretinal disorder, prompted us to obtain ornithine levels on samples of blood and urine.2-4 Serum and urine ornithine levels were normal. Significantly elevated ornithine levels would have strongly suggested a relationship between pigmented paravenous retinochoroidal atrophy and gyrate atrophy. Little is known about the early fundus features of gyrate atrophy,5 despite the occasional occurrence of the entity as an autosomal dominant disease. Likewise, little is known about the late stages of pigmented paravenous retinochoroidal atrophy.

In advanced gyrate atrophy, colorless, elongated, glittering crystals have been described.³ Although our patient showed similar crystal deposition, we do not relate the two diseases because the scalloped lesions of gyrate atrophy are typically peripheral and not posterior, as in this case. Elevated ornithine levels further distinguish gyrate atrophy from pigmented paravenous chorioretinal atrophy.

SUMMARY

A 30-year-old man with pigmented paravenous chorioretinal atrophy showed, within a relatively short time, changes that documented the progressive nature of this disease. These changes included: further constriction of peripheral visual fields; more extensive and frequently confluent areas of retinochoroidal atrophy; a scalloped appearance of lesions resem-

bling posterior gyrate atrophy; peripheral pigment clumping; and the presence of localized atrophic areas with crystal deposition in the peripheral retina.

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OPHTHALMIC MINIATURE

If a statistically-minded physician were to go through his notes and tabulate the various symptoms of which his patients complained, and were then to place these in order of frequency, he would find that the commonest ill from which his patients suffered was fear. Having discovered this he might proceed to classify the types of fear which were responsible for his patients coming to consult him. He would find that sometimes the fear was the cause, and sometimes the result of their illness. Sometimes the patient was aware of it, and sometimes entirely unconscious of the explanation of his suffering. Sometimes the fear revealed itself in the preliminary remarks with which the patient opened the conversation, and sometimes it only became obvious as he was about to leave.

Kenneth Walker, *The Circle of Life* Johnathan Cape, 1942

PUNCTATE LENTICULAR OPACITIES IN TYPE II MANNOSIDOSIS

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Mannosidosis is a recently recognized. inborn error of metabolism1 resulting from the deficient activities of acidic αmannosidases A and B.1,2 It is characterized by the lysosomal accumulation of mannose-rich glycopeptides, glycoproteins, and oligosaccharides in tissues and fluids of affected individuals.1,3-7 The clinical man festations of this lysosomal storage disease include mild to moderate psychomotor retardation, a facial dysmorphia resembling that seen in the Hurler syndrome, dysostosis multiplex, hepatosplenomegaly, sensorineural hearing loss, corneal or lenticular opacities, susceptibility to infections, and autosomal recessive inheritance.1.8 The phenotypic resemblance of patients with mannosidosis to these with the mucopolysaccharidoses has presumably often led to misdiagnosis in the past.

The ocular findings in homozygous patients with mannosidosis have been described previously. These abnormalities include: (1) lenticular changes characterized by posterior cortical opacities in a spoke- or wheel-like pattern; (2) corneal changes consisting of superficial opacities; (3) slight pallor or graying of the optic disk, as well as blurring of the disk margins; and (4) esotropia.

Recently, two clinical subtypes of mannosidosis have been recognized based on differential clinical and biochemical findings.11.12 Type I homozygotes have severe disease with psychomotor retardation, short stature, dysostosis multiplex, hepatosplenomegaly, severe recurrent infections, defective chemotaxis, and early death. Type II homozygotes have a milder course, characterized by moderate mental retardation, almost normal stature, milder dysostosis multiplex, hearing loss, and survival into adulthood (Table 1). To date, we have examined five homozygous patients with mannosidosis. Four homozygotes (Cases 2-5), with Type II mannosidosis, ranging in age from 16 to 25 years old, were the offspring of parents who were first cousins; the other homozygote with Type I (Case 1) disease was unrelated and has since died at age 21/2 years. The clinical and biochemical findings in these patients have been described elsewhere. 11 We report here the ocular findings in these patients and, in particular, the lenticular lesions observed in all four Type II homozygotes. The finding of these punctate opacities randomly scattered throughout the entire lenticular cortex may provide a further clinical distinction of variants of mannosidosis.

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CASE REPORTS

Case 1—This 2-year-old girl was examined on Aug. 30, 1973. Examination of visual acuity demonstrated the ability to fix and follow large objects and light readily with either eye. Slit-lamp examination revealed clear corneas, lenses, anterior chambers, and normal irides. External examination showed telecanthus with an intrapupillary distance of 52 mm; examination of the pupils and motility was normal. Cycloplegic examination revealed normal disks with 0.2 cup:disk ratio in both 6, es; the optic disks were slightly elliptical in the vertical direction, but no other abnormalities were obserted.

TABLE 1 DIFFERENTIAL PHENOTYPIC MANIFESTATIONS IN MANNOSIDOSIS TYPES I AND II

Manifestations	Type I	Туре П
Psychomotor retardation	Moderate-severe	Mild-moderate
Short stature		+1-
Facial dysmorphia		
Dysostosis multiplex	Severe	Mild
Hepatosplenomegaly	+ 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1	
Lenticular opacities	그 그 그 그 아이를 가셨다면 하셨다.	+/-
Corneal opacities	+/-	**************************************
Hearing loss	+	* * *
Recurrent infections	+ Barrier 1984	
Chemotactic defect	+	
Decreased serum IgG	+ Addin 1997	\
Survival	Early death	Adulthood

Case 2-This 25-year-old white man is the sibling of homozygotes in Cases 3, 4, and 5; he was examinined on May 10, 1976. Visual acuity without correction was 6/9 (20/30) in both eyes assessed with the Snellen illiterate E game. Examination of the pupils and extraocular muscles revealed no abnormalities. Intraocular pressures were normal. Cycloplegic examination revealed refractive errors of R.E.: +4.50 $+1.00 \times 80$, and L.E.: $+3.00 +1.00 \times 100$. The corneas, anterior chambers, and irides were normal on slit-lamp examination. However, both lenses revealed scattered punctate opacities (Fig. 1), additionally, the left lens had an anterior plaque-like opacity, in the superior temporal quadrant, covering approximately 25% of the dilated pupil (Fig. 2). The fundi were normal.

Case 4—This 18-year-old white man was examined on May 5, 1976. He was able to fix and follow light readily with either eye. Results of external examination, including the pupils and extraocular muscles, were normal in all respects. Slit-lamp examination revealed no abnormalities of the corneas, anterior chambers or irides; however, scattered punctate opacities were observed throughout the lens in both eyes. Ocular pressures determined by digital palpation seemed normal. Cycloplegic examination revealed refractive errors of R.E.: +0.50 +0.75 × 70, and L.E.: +1.25 sphere. The optic disks had a slight pallor and there was a slight blurring of the optic disk margins; the remainder of the ophthalmoscopic examination was normal.

Case 5—This 16-year-old white girl was examined on May 10, 1976. Visual acuity without correction was 6/9 (20/30) +2 bilaterally. Pupils, extraocular muscles, and results of external examination were normal. Slit-lamp examination revealed normal corneas, anterior chambers, and irides. The lenses contained punctate opacities randomly scattered throughout the cortex. Cycloplegic examination revealed a refractive error of R.E.: +1.25 sphere, and

L.E.: $+0.75 +0.50 \times 90$. The disks were normally colored with blurred margins, otherwise the fundit were normal.

DISCUSSION

We reviewed the ophthalmologic findings in the 37 homozygous—patients with mannosidosis reported as of July 1977, as well as the findings in our five

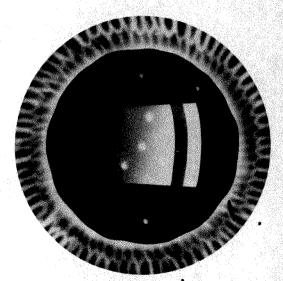


Fig. 1 (Letson and Desnick, Typical slit-lamp microscopy of the lenticular opacities in Cases 2 to 5 with Type II mannosidosis. These small punctate opacities with indistinct margins were distributed throughout the lens.

TABLE 2
OPHTHALMOLOGIC FINDINGS IN HOMOZYGOTES WITH WANNOSIDOSIS

	1		Age at	Pre-	-	On the lands of Finding	Tinding.	
Case			Report	sumed		Communication of the state of t	* maings	
So.	Reference	Sex	(yrs)	Type	Lens	Con ea	Fundus	Other Comments
•	Aylsworth and associates 13	Z	က	Ţ	Spoke-like cataracts	<i>!</i>		
Ø	Arbisser and associates ⁹	Σ	ıo	-	Posterior spoke-like vacuolar spacities	70.	14	nl intraocular pressure; poor vision, left eye, -8.00 both eyes;
ಌ		L .	22/5	-	Posterior spoke-like vacuolar opacities	n.	Ē	estropia n1 intraocular pressures;
4		6	310/12	<u> </u>	Mild posterior spoke- like vacuolar onacities	- Tu		ni EKÇ
າດ	Autio and associates ¹⁴	×	910/12		0.1			Esotropia
∽ 1 ∞	Bach	ZZZ	3 ¹ / ₂ 2 ¹⁰ / ₁₂ 11		Wheel-like lens opacities Wheel-like lens opacities			
6 ² 1	and associates ²⁰ Booth, Chen, and Nadler ¹⁷	[24 [24	5 26	ĦĦ	- Tu	Mild opacities n1	• 10	Refractive error
11 12 13	Farriaux	ZZZ	16 13 4	III	Tu Tu Tu	72 73	3	Refractive error
≖⊭	and associates ¹⁸		41/2	â:				
192		Z 14 3	12*/2 10	3 2 2	-			
18	Loeb and associates 19	ž Œ	13	.				
28	Norden, Ockermann,	M	7.	=-		nd Superficial opacities		
	end Stabbo							

TABLE 2 (Continued)
OPHTHALMOLOGIC FINDINGS IN HOMOZYGOTES WITH MANNOSIDOSIS

Type Lens Cornea			Age at	Pre-		Ophthalmologic Findings	gic Findings	
F 4 1		Sex	Report (yrs)	sumed Type	Lens	Cornea	Fundus	Other Comments
Spranger, F 6 II Small cloudy patches n 1 1 1 1 1 1 1 1 1	21	4	**	-		Moderate superficial		
Spranger, Cohler, and Cantz/18 F 6 II Ontanterior surface Cohler, and Cantz/18 F 9 III n l P		X	4"/3		Small cloudy patches	opacities n1	Disks gray and	
Cohlor, and Cantz ¹⁵ F 9 11 11 11 11 11 11		4	9	Ħ	on antenor surrace n1			
F 9 11 n1	Gehler, and Cantz ¹⁵							
F 17/3 P n F 6 1 Cataract M 2 17 n F 6 P n F 9 11 n M 10 11 n F 13 11 n F 13 11 n Kistler M 26 P n Present cases F 21/2 I Scattered punctate F 21 11 Scattered punctate F 16 11 Scattered punctate F 17 Scattered punctate F 18 19 11 Scattered punctate F 19 11 Scattered punctate F 10 11 Scattered	24 70	<u> </u>	6 -					
F F F F F F F F F F	98	, E4 F	11/3	ا م	Te :			
M 12 13 14 15 15 17 18 18 18 18 18 18 18	28 28 38	L Z	ဝ းဂ	- 61	Cataract			
F 9 11P 11 11 11 11 11 11	50	⊠ ta	12	ei e	Tu Tu			
M 10 11 11 11 11 11 11	31	. II.	ာတာ	eii	l'a			
F 13 II	32 33	ΣĿ	9 =	= =	-			
Kistler M 26 11 nl nl and associates ²¹ M 6 ? nl nl Present cases ¹¹ F 2 ^{1/2} 1 Scattered punctate nl cortical opacities; M 19 II Scattered punctate nl cortical opacities M 19 II Scattered punctate nl cortical opacities F 21 II Scattered punctate nl cortical opacities K 21 II Scattered punctate nl cortical opacities	7.	£.;	13	II S	1.0			
and associates ²¹ M 6 7 n1 n1 n2 n2/2 11 Scattered punctate n1 cortical opacities, anterior plaque cortical opacities R 21 11 Scattered punctate n1 cortical opacities M 19 11 Scattered punctate n1 cortical opacities n2 cortical opacities n2 cortical opacities n3 cortical opacities n4 Scattered punctate n6 Scattered punctate n7 Scattered punctate n6 Scattered punctate n7 Scattered punctate n7 Scattered punctate n1			36	=	nl	nı	•	
Present cases 11 F 21/2 I n1 n1 n1 n1 cortical opacities; anterior plaque recording to the cortical opacities n1 cortical opacities n2 cortical opacities			g	o.	Tu	[u		
M 26 II Scattered punctate nl cortical opacities; anterior plaque F 21 II Scattered punctate nl cortical opacities M 19 II Scattered punctate n cortical opacities F 16 II Scattered punctate nl			21/2	-	nl	Tu	Tu	See text
F 21 II Scattered punctate n1 cortical opacities M 19 II Scattered punctate n cortical opacities F 16 II Scattered punctate n1			26	П	Scattered punctate cortical opacities; anterior plaque	Ta .		See text
M 19 II Scattered punctate n cortical opacities F 16 II Scattered penctate n1	• 07	<u>Da</u>	7	=	Scattered punctate cortical opacities	nl	of disks, blur- ring of disk	See text
F 16 II Scattered penctate	4	Z	61	1	Scattered punctate cortical opacities	·	Slight pallor of disks, blurring	See tex
	422	Œ.	16	-	Scattered penctate cortical opacities	Ţ	Blurring of disk margins	See text

* - indicates not reported.
in indicates normal findings.



Fig. 2 (Letson and Desnick). Direct illumination photograph of the left lens in Case 2 with Type II mannosidosis. Note the anterior placque-like opacity in the superior-temporal quadrant.

patients (Table 2). These patients were classified as Type I or II mannosidosis on the basis of the clinical and laboratory findings in each case. Older patients presumably represent Type II homozygotes because of their milder clinical manifestations, lack of severe recurrent infections, and reported survival to adulthood. More severely affected patients presumably represent Type I disease.

Of the patients whose lenses were examined, eight patients, all classified as presumed Type I variants, had lenticular changes; six of these were characterized as opacities in a spoke- or wheel-like pattern, three were posterior, 9,13 and three unspecified14,15 in position. Additionally, one presumed Type I patient had cloudy appearing opacities on the anterior surface.1 No lenticular changes were observed in the 17 presumed Type II patients whose lenses were examined and reported.15-17 In contrast, of the four siblings with Type II mannosidosis described in this report all had punctate lenticular opacities scattered randomly throughout the entire lens (Fig. 1). This distribution of the opacities is consistent with the finding that α-mannosidase activity is distributed uniformly in anterior

to posterior sections of the human lens, in contrast to several other lysosomal hydrolases studied. 16

Of the 15 patients whose corneal findings were recorded, corneal opacities were described as superficial in two presumed Type I homozygotes³ and mild in one Type II patient²⁰; the other 13 patients reportedly had clear corneas. None of the five presently reported cases had corneal changes. Inadequate information was available for comment on the ophthalmoscopic, refraction, strabismus, and visual acuity findings in these patients.

The lenticular findings in previous studies occurred in presumed Type I homozygotes. However, it is not possible to distinguish definitively severe Type I mannosidosis from the milder Type II disease by ophthalmologic examination. in young children. For example, our Type I homozygote did not have lenticular or corneal changes at 21/2 years of age. Perhaps continued study of these patients will provide clinical evidence for the hypothesis that Type I patients can be distinguished by the presence of posterior lenticular opacities in a spoke-like pattern, whereas the Type II homoxygotes may have no lenticular involvement even

in their late 20s.¹⁷ Support of this concept would provide valuable prognostic information for the families of patients with newly diagnosed cases. Alternatively, the lenticular lesions may result from the slow, but progressive accumulation of mannose glycoconjugates in the lens, thus accounting for this finding in older patients, as well as more severely affected young patients, no matter what subtype.

Genetic heterogeneity in mannosidosis has been suggested by several investigators based on the remarkably variable severity of the clinical manifestations 11,13,17,20 As expected for rare recessive diseases, unrelated homozygotes presumably represent genetic compounds of two different allelic, recessive mutations.22 The occurrence of multiple mutant alleles at a particular gene locus can be the source of genetic heterogeneity, as has been shown for the Hurler/Scheie variants with defective α-iduronidase activity.23 In mannosidosis, the variation in clinical manifestations from mild17,20 to severe^{5,13} disease, may result from different allelic mutations of the structural genes responsible for lysosomal α-mannosidase A and B activities.13 In this disorder, most patients have residual activity of the defective enzymes (3 to 20% of normal activity); different mutations may result in various rates of mannoseglycoconjugate degradation and accumulation in vivo and, consequently, different expressions of clinical severity. Patients with relatively more α-mannosidase activity or more stable enzymes may have distinctly milder clinical manifestations, as exemplified by the mild clinical involvement and approximately 20% residual activity in the two siblings reported by Bach and associates.20 In addition to the differential phenotypic manifestations, the Type I and Type II patients described here were biochemically differentiated by the relative cryo- and thermostabilities of their respective residual amannosidase activities.¹² Thus, all patients with mannosidosis should have a thorough historical and clinical evaluation, including a complete ophthamologic examination and appropriate chemical and enzymatic assays. These studies may allow assignment of a subtype which would provide important prognostic information for optimal patient management and family counseling.

SUMMARY

Ocular examination in four siblings with Type II mannosidosis revealed scattered punctate opacities in the antire lens. No such lenticular opacities were described in 28 previously reported patients, 17 presumed Type II and 11 Type I. Complete ocular examination with particular attention to opacities in the lens and cornea may provide additional phenotypic data for identification of genetic variants with mannosidosis.

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OCULAR BIOAVAILABILITY AND SYSTEMIC LOSS OF TOPICALLY APPLIED OPHTHALMIC DRUGS

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The bioavailability of topically applied pilocarpine nitrate as a function of instilled volume has been reported. By using a pharmacokinetic treatment, this study showed that as the instilled volume was decreased, the fraction of dose absorbed into the interior of the eye increased. Since drainage of instilled solutions into the nasolacrimal duct is a function of instilled volume, the potential for systemic loss of drug should also be minimized by administering doses in volumes smaller than are commonly used.

When a drug is applied locally to an area of the body, the intent is to affect the immediate area of application. Topically applied ophthalmic drugs are intended to exert some local effect or to penetrate the cornea and act on some internal ocular structure. Loss of drugs to other areas of the body and particularly into the general systemic circulation could cause toxic side effects. Such systemic drug loss could be especially harmful in both pediatric and geriatric patients because these age groups may be less able to tolerate large doses of systemically absorbed drug.^{3–5}

Much effort in recent years has been devoted to evaluating topical ophthalmic arug delivery in an attempt to optimize

ocular bioavailability. The development of solid drug delivery devices^{6–12} has been a major advance in this direction. However, the instillation of drugs in drop form continues to be the major method of applying drugs to the eye. Therefore, systematic examination of the factors responsible for drug distribution after this form of drug delivery is essential to place ophthalmic dosage regimens on a more actional basis.

Most investigators have concerned themselves with vehicle influences on drug penetration.^{13–19} In this study, our aim was to maximize drug concentration in the eye while simultaneously minimizing systemic drug loss.

MATERIAL AND METHODS

Commercially obtained pilocarpine nitrate USP was used without further purification. Tritiated pilocarpine (specific activity 4.1 Ci/mM) in ethanol solution was evaporated several times before use²⁰ to remove any solvent that had become tritiated by exchange. All other chemicals were of analytical or reagent grade and were used as received.

All rabbits used were male New Zealand albinos, 18 to 23 days old. Before experimentation, rabbits were housed in standard laboratory animal cages and allowed food and water at will.

Pilocarpine nitrate solutions were prepared in isotonic Sorensen's phosphate buffer at a pH of 6.24. Solutions were filtered for clarity but not sterilized, and were prepared fresh for each experiment. The amount of tritiated pilocarpine added was chosen to insure adequate counting accuracy, but in no case was it sufficient

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to alter the molarity of the final solutions. Final solutions yielded a counting activity of approximately 60,000 counts per minute per microliter of solution.

Pilocarpine concentrations in both aqueous humor and plasma were determined by tritium measurement in a liquid scintillation spectrometer, with 5 ml of a commercial scintillation cocktail in polyethylene vials. Counting efficiencies were constant for all samples on the basis of the external standards ratio. We used suitable standards and blank corrections to convert counts to micrograms of drug per milliliter of plasma or aqueous humor.

Acatous humor concentration-time nrofiles-Rabbits were weighed and placed in restraining boxes in the upright position. Head movement was restricted, but normal eye movement was maintained. We delivered the appropriate volume and concentration of pilocarpine solution to the eye by using a microliter syfinge. At various times after instillation. rabbits were killed with an overdose of pentobarbital sodium, eyes were thoroughly rinsed and blotted, and aqueous humor was aspirated from the anterior chamber. In each case, 55 to 75 µl of aqueous humor was removed, and 50 µ1 was accurately transferred to counting vials in which the previously described cocktail had been prerefrigerated for at least 24 hours. Samples were mixed and stored in the dark at room temperature for at least 24 hours before counting. For each concentration, samples were taken at five, ten, 15, 20, 30, 45, 60, 90, and 120 minutes after instillation. Six to eight samples at each point were obtained.

Plasma concentration-time profiles— Animal positioning and drug instillation were the same as described above except that instillation was made into only one eye of each rabbit. At specified times after instillation (15, 30, and 60 minutes) rabbits were killed and 2.5 ml of blood was withdrawn into a heparinized syringe via cardiac puncture. The blood was transferred to a 5-ml styrene culture tube agitated gently, and centrifuged for ten minutes at 2200 rpm, 0.5 ml of plasma was added to the scintillation vial and treated as previously described. Again, the use of suitable standards and blank correction permitted conversion of counts to micrograms of drug per milliliter of plasma. Obviously, with the analytical method used here no distinctions could be made between intact and metabolized drug, so all values reported refer to total radioactivity.

RESULTS

Theoretically, in 60-day-old rabbits, a 5 μ l dose of 1.61 \times 10⁻²M pilocarpine nitrate should result in the same area under the aqueous humor concentration-time profile as a 25 μ l dose of 1.00 \times 10⁻² M pilocarpine nitrate. This calculation is based on the finding that as the instilled volume is decreased, the fraction of applied dose that is absorbed increases. The validity of this calculation has recently been confirmed. ²¹

Using 20-day-old rabbits as a model for ocular drug penetration in the immature eye, a similar argument can be developed. A 5 μ l dose of 1 \times 10⁻²M pilocarpine nitrate was administered to the eyes of 20-day-old rabbits and the aqueous humor concentration-time profile constructed. The results of this study are shown (Table). By using the trapezoidal rule with extrapolation to infinity, we calculated the area under concentration-time profile to be 59.6 µgmin. ml⁻¹ (using a terminal slope of 2.80 × 10⁻²min⁻¹). With these data and the pharmacokinetic one compartment open model, it is possible to calculate the fraction of applied dose absorbed according to the following equation:

$$F = \frac{(A) (V) (K)}{D} \text{Equation (1)}$$

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TABLE

AQUEOUS HUMOR CONCENTRATION-TIME PROFILE FOLLOWING INSTILLATION OF $5~\mu 1$ OF 1×10^{-2} M PILOCARPINE NITRATE IN THE EYES OF 20-DAY-OLD RABBITS

Minutes	μg Pilocarpine/ml Aqueous Humor*
5	0.46 (.03)
10	0.84 (.14)
15	1.04 (.12)
20	1.42 (.15) 1.09 (.19)
30 45	0.56 (.06)
60	0.28 (.02)
90	0.13 (.01)
120	0.10 (.02)

^{*}Mean concentration, with standard error in parentheses. Each point is the mean of at least eight determinations.

where F is the fraction of dose absorbed, A is the area under the concentration-time profile, V is the volume of distribution, K the elimination slope, and D the applied dose. Assuming V to be the aqueous humor volume and using the reported value of 0.127 ml for 20-day-old rabbits, 22 F is calculated to be 1.56×10^{-2} .

When 25 μ 1 of 1 × 10⁻²M pilocarpine nitrate is adminstered to 20-day-old rabbits, previous data³ reveal an area under the aqueous humor concentration-time profile of 114.5 μ g-min ml⁻¹. We can, therefore, calculate what dose administered in 5 μ 1 should result in the same

area under the curve as a 25 μ 1 dose of 1 \times 10⁻²M pilocarpine. Rearranging equation 1;

$$D = \frac{(A)(V)(K)}{F}$$
 equation (2)

and substituting in the appropriate numbers (A=114.5, V=0.127, $K=2.80 \times 10^{-2}$ and $F=1.56 \times 10^{-2}$), D is calculated to be 26.1 μ g. This corresponds to a solution concentration of 1.92×10^{-2} M pilocarpine nitrate administered as a 5- μ 1 drop. In other words, a 5- μ 1 drop of 1.92×10^{-2} M pilocarpine nitrate a predicted to give the same area under the aqueous humor concentration-time profile as a 25- μ 1 drop of 1×10^{-2} M solution. We recorded comparative aqueous humor profiles (Fig. 1) and comparative plasma level profiles (Fig. 2).

DISCUSSION

For the 5- μ 1 dose of 1.92 \times 10⁻²M pilocarpine, the area under the aqueous humor concentration-time profile is calculated to be 113.9 μ g min ml⁻¹, in excellent agreement with the predicted value of 114.5 μ g min ml⁻¹. The administered dose has been reduced from 67.8 μ g to 26.1 μ g, a factor of 2.6 times without loss of drug concentration in the aqueous humor. These results imply that the volume of drug solution normally delivered by commercial ophthalmic droppers

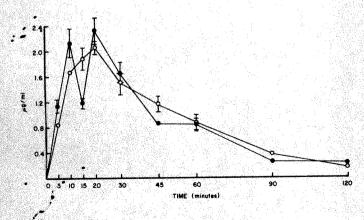


Fig. 1 (Patton and Francoeur). Aqueous humor concentration-time profile of pilocarpine after instillation of 25 μ l of a 1 \times 10⁻² M solution (open circles) and 5 μ l of a 1.92 \times 10⁻² M solution (solid circles) in 20-day-old rabbits.

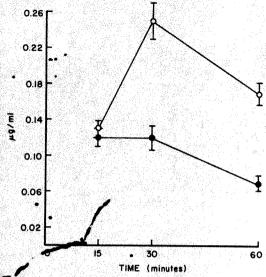


Fig. 2 (Patton and Francoeur). Plasma concentrations after instillation of 25 $\mu 1$ of a 1×10^{-2} M solution (open circles) and 5 $\mu 1$ of a 1.92×10^{-2} M solution (solid circles) of pilocarpine into the eyes of 20-day-old rabbits.

(usually 50 to 75 μ 1) may be excessive and not necessary to achieve the desired drug levels in the eye. The finding that the fraction of dose absorbed into the eye increases as the instilled volume is decreased indicates that more efficient topical drug delivery can be achieved by administering doses in volumes smaller than currently used.

Futhermore, the plasma level profiles indicate that considerably larger quantities of drug appeared in the blood after the 25 μ l drop of 1×10^{-2} M pilocarpine nitrate than the 5 μ l drop of 1.92×10^{-2} M. This is to be expected because the dose in the former case is larger. However, because aqueous humor levels of drug have been maintained with the smaller dose while blood concentrations are lower also argues strongly for the development of better small volume delivery devices for ophthalmic drugs.

Both infants and elderly patients are less likely to tolerate drug concentrations

resulting from systemic absorption after topical application to the eye. The potential for adverse side effects, therefore, is a concern. In the particular case studied. with the $5 \mu 1$ drop (26.1 μg) the fraction absorbed into the eye (F) was 1.56×10^{-2} or 0.41 µg. This leaves 25.7 µg potentially available for systemic absorption. A similar calculation on the 25 µ1 drop (67.8 µg) shows an absorbed fraction of 5.28×10^{-3} or 0.36 µg, thus leaving 67.4 µg potentially available for systemic absorption. Assuming substantial or complete systemic absorption of this dose, unwanted side effects are clearly more likely with the larger drop size.

An understanding of the factors responsible for the distribution and movement of drugs after topical application to the eye can lead to more rational, effective, and safer delivery via this route. The advantages of reduced drop sizes for topical ophthalmic drug delivery are twofold; first, smaller doses can be administered without losing drug concentration in the eye; and second, such doses result in less systemic drug loss.

SUMMARY

We used 20-day-old rabbits as a model to show that the ocular bioavailability of topically applied pilocarpine nitrate increased as the instilled volume of the drug was decreased. Decreasing the instilled volume from 25 to 5 µl permitted a dosage reduction of greater than 2.5 times without sacrificing overall drug concentrations in the eye. Since only a small. fraction of topically applied doses to the eye actually reached the interior of the eye, the remainder of the dose was lost and available for systemic absorption, The reduction in dosage permitted by this approach resulted in less drug appearing in the general circulation, as shown by comparative plasma level-time profiles. The advantages of reducing drop size are improved ocular bioavailability permitting the use of smaller doses; and less systemic drug loss, thus reducing the potential for systemic side effects. These advantages could be especially significant in the pediatric and geriatric age groups.

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SIMULTANEOUS STEREOPHOTOGRAMMETRIC AND ANGIOGRAPHIC FUNDUS CAMERA

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Since the development of fluorescein fundus angiography in 1960, we have recognized the importance of stereoscopic photography in permitting the three dimensional observation of various ocular fundus pathologies. Allen's corneal induced parallax method1-3 is the most widely used in the stereo fundus photographic field and has been applied to fluorescein angiography. However, this method uses approximate rather than absolute stereo photography. That is, subsequent paired exposures are obtained to produce a pair of stereo photographs. This lack of simultaneity generates charfges in the photographed field and a variability of metric stereoscopic base, thus making accurate stereophotogrammetric analysis impossible.

As early as 1930, Nordenson⁴ produced simultaneous stereoscopic fundus photographs with a specially constructed Zeiss-Nordenson camera. These photographs were obtained by a pair of small prisms mounted in front of the camera lenses. In 1953 H.J. Norton⁵ made stereoscopic fundus photographs by attaching a 35-mm camera to a Bausch and Lomb binocular ophthalmoscope. In 1957 Drews⁶ took simultaneous stereoscopic fundus photographs by using the principle of indirect

ophthalmoscopy. None of these methods has been entirely satisfactory because of the small depth effect obtained, the lack of good photographic resolution, and the presence of illuminating light source reflexes superimposed on the fundus image.

In 1965 Donaldson⁷ developed a simultaneous stereoscopic fundus camera. This camera, composed of two separate optical systems, provided excellent stereoscopic fundus photographs consisting of two physically separated 35-mm frames. However, at that time, the illumination system used did not allow for fluorescein angiography and the camera was difficult to manipulate.

Recently, Saheb, Drance, and Nelson⁸ and Schirmer and Kratky⁹ introduced twin-prism methods for simultaneous stereoscopic fundus photography. They hoped to photogrammetrically detect and document progressive changes of optic disks in glaucoma. However, the prisms have added a distortion to the optical system, thus impairing proper stereophotogrammetry and reducing the photographed field.

An accurately documented comparative study¹⁰ of the photogrammetric error generated by the Allen corneal-induced parallax method, the biprism method, and the Donaldson simultaneous fundus camera showed the clear superiority of the latter. The Donaldson simultaneous fundus camera was not commercially produced and thus not made available to the ophthalmological community. This lack has greatly hindered research on the morphological changes of the retina and, in particular, on glaucoma and its treatment.

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A simultaneous stereophotogrammetric fundus camera should produce paired stereoscopic images in a single 35-mm frame during fluorescein angiography. It should also allow for ready mounting of paired stereoscopic images and filing of these photographs.

Furthermore, one should be able to observe these stereoscopic photographs with any commercially produced, single-frame stereoscopic viewer. Finally, the camera should be easily manipulated.

Matsui and co-workers¹¹ reported the first such model, a modified Topcon TRC-F fundus camera, about two years ago. With this camera, pairs of stereoscopic angiographs gave satisfactory results in picturing pathologically elevated lesions as seen in fundi with choked disks, but the findings were unimpressive when applied to the normal fundus. This was because of the insufficient value of the metric stereoscopic base at the plane of the subject's pupil.

We have recently developed a second type of simultaneous stereoscopic fundus camera* that can be applied to serial fluorescein angiography, and thus overcomes the disadvantages of the first type. This camera provides excellent stereoscopy of both the normal fundus and the fundus with pathological lesions; it is particularly suited for accurate photogrammetric studies of the human fundus.

METHODS

A side view of the apparatus (Figs. 1 and 2) and an optical diagram of this system (Fig. 3) are shown. The principle of the system is explained schematically (Fig. 4). The inverted image focused through an aspheric objective lens of a regular Topcon fundus camera is separated horizontally into a pair of images by a beam-

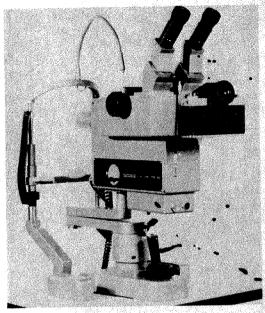


Fig. 1 (Matsui, Parel, and Norton). Rear view of the apparatus showing the binocular viewing tube and the monocular photographic eyepiece.

splitter placed at the plane conjugate to the plane of the subject's pupil. The beam-splitter is made up of a pair of Poro prisms. The Poro prism inverses the image vertically and reverses it horizontally. The paired stereoscopic pupils placed in front of the Poro prisms provide either a 2.5-mm or 3.0-mm stereoscopic base and theoretically require the subject to have a 6-mm pupil. The pair of stereoscopic images made by the beam-splitter are focused on a single frame of 35-mm film through a pair of relay lenses and a pair of prisms. The elapsed time of the dye injection is also documented in the same frame (Fig. 5).

An oval-shaped mask is placed in the illuminating pathway to create the special shape of an unilluminated cone in the subject's anterior segment to eliminate reflexes or artifacts on the photograph (Fig. 6). The power supply unit of this system permits one exposure a second so that serial, simultaneous stereoscopic

^{*}This instrument is available from Topcon Instrument Corp. of America, 9 Keystone Place, Paramus, NJ 07652.

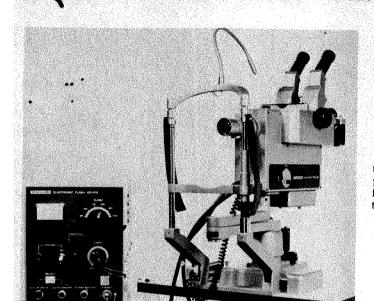


Fig. 2 (Matsui, Parel, and Norton). Frontal view. The power supply for simultaneous stereoscopic fundus angiography is displayed at the left.

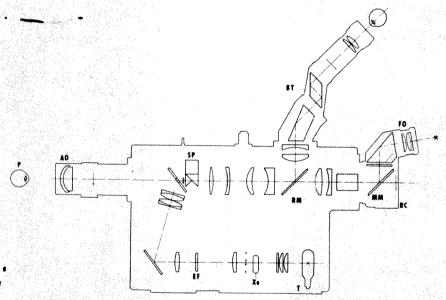


Fig. 3 (Matsui, Parel, and Norton). Optical diagram of the apparatus. T designates tungsten bulb; x_e , xenon flash lamp; EF, exciter filter; AO, aspheric objective; SP, separation prisms and stereobase; RM, removable mirror; MM, motorized mirror; RC, recording camera back with barrier filter; FO, focusing ocular; BT, binocular stereoscopic tube.

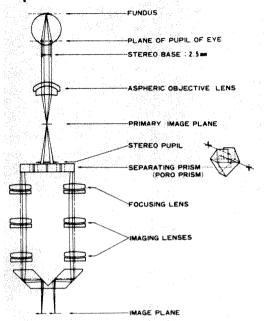


Fig. 4 (Matsui, Parel, and Norton). Principle and ray tracing of the imaging stereoscopic system.

photographic angiography can be performed. With the turn of a knob, an exciter filter (Spectro-tech SE40) is inserted in the illuminating pathway and a barrier filter (Spectro-tech SB50) is placed in front of the film. Transmission characteristics of these filters are shown (Fig. 7). Color and red-free simultaneous stereo-

scopic photographs can also be produced by this unit.

To ease the photographer's burden, a motorized film drive pushbutton control has been installed on the joystick to control the fundus camera's lateral movement. A binocular stereo tube allows the photographer to survey the fundus in three dimensions, thus facilitating the recognition of retinal details. This binocular tube has an adjustable interpupillary distance of 50 to 80 mm and magnifies the fundus 16 times. For accurate stereophotography, the focusing is done through the monocular eveniece of the motorized camera back in which a graticule, equidistant to the film plane, has been mounted. The switchover between binocular stereoscopic and monocular viewing is performed by a movable mirror.

For photogrammetric purposes, the photographer chooses the stereoscopic camera base in advance. A 3-mm base would be used when large pupillary dilatation can be obtained; a 2.5-mm base is usually chosen for pupils of about 6 mm in diameter. The exact stereoscopic base value is important for the stereophotogrammetric reconstruction of the patient's fundus. The optical system has a constant angular magnification of × 2.56, regardless of the patient's refractive error. That

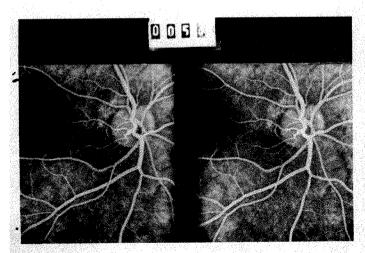


Fig. 5 (Matsui, Parel, and Norton). Paired stereoscopic angiograms of a 30-year-old man taken 5.3 seconds after fluorescein injection.

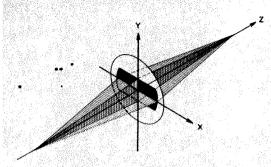


Fig. 6 (Matsui, Parel, and Norton). Ray tracing of the shape of unilluminated cone at the subject's pupil.

particular magnification was chosen to photograph the parent's optic disk and his macula on a single 16 × 20-mm frame.

The linear magnification is a function of the patient's refractive error and can be computed with the following equation:

$$M_t = (43.594/f_s) \pm 0.035$$

where f_s is the patient's eye reduced focal length expressed in millimeters. For the Gullstrand schematic eye, $f_s \approx 17.05$ -mm, thus $M_I \approx 2.56 \pm 0.035$. The error value given here is valid for photogrammetric comparison performed on two different cameras. When using the same camera, this error is constant and thus does not affect the stereophotogrammetric computations of the patient's fundus morphology.

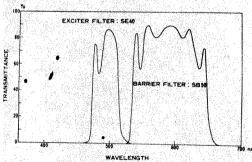


Fig. 7 (Matsui, Parel, and Norton). Spectral transmittance characteristics of the exciter and barrier filters used for fluorescein stereoscopic angiography.

In theory, 6 mm of pupillary dilatation is required; however, more fully dilated pupils yield the best results.

Color photographs are always taken before fluorescein angiography with an Ektachrome type color film and a flash intensity of 100 joules. For fluorescein angiography a Tri-X type of black and white film is used and the flash intensity is increased to 300 joules (watt-second), Forced development is required which approximates ASA 1,000.

Five milliliters of 10% fluorescein sodium solution is injected rapidly into the subject's antecubital vein. Twenty pictures are taken continuously at one-second intervals and several more pictures at five-minute, 15-minute, and if necessary, at 20- to 30-minute intervals. The film was developed in Konidor Super for about 15 minutes at 24° C, or in D11 for seven minutes.

Observation of the stereoscopic angiograms is made through a single frame stereo viewer (Fig. 8).

RESULTS

The stereoscopic photographs taken by the camera gave satisfactory results in young persons even though they had only 4-to 5-mm dilated pupils. In older persons the results were less impressive if their pupils were not well dilated.

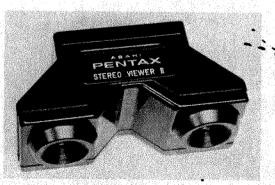


Fig. 8 (Matsui, Parel, and Norton). Single frame-stereoscopic viewer.

The forced development technique produces a moderate granularity of the fluorescein angiographs. However, we can easily recognize capillary changes three dimensionally through a stereoscopic viewer. A serial angiograph taken by this camera is shown (Fig. 9). The stereoscopic angiograms of this series provided excellent stereoscopy.

Discussion

There are a number of advantages in this system. One can obtain paired stereoscopic photographs in a single 35-mm frame, which greatly simplifies mounting and filing. Serial simultaneous stereoangiograph can be performed easily at the push of a button. Observation through a stereoviewer always permits satisfactory stereoscopy. Accurate stereophotogrammetric analysis of the fundus can be performed. Each picture of the stereoscopic pair has a rectangular shape of 16×20 mm. To simultaneously encompass the

optic disk and the macula on a single frame, the optical magnification was set at 2.56. Therefore, each picture covers almost the same area as a fundus photograph taken with a standard fundus camera. To survey the retina, observation through the eve piece is binocular, and the observed images are seen in three dimension. The inverted images in the monocular eve piece give some difficulty when photographing, although this is easily overcome with practice. However, ... the inverted images on film are convenient for mounting because they do not need to be separated. We can observe erect images easily by turning each stereo frame upside down (Fig. 9).

There are many applications for this camera. One of the most important is documentation of disk cupping in glaucoma studies because serial comparison of cupping grade can be performed as a result of the constant stereo base. Kottler, Rosenthal, and Falconer recently re-

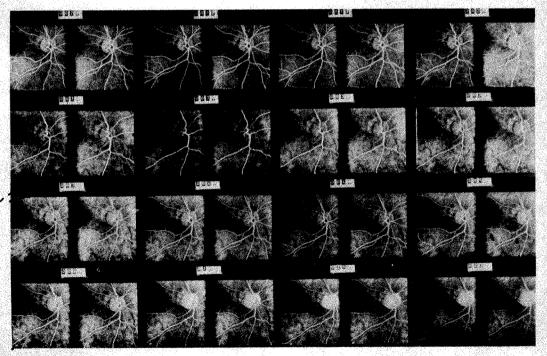


Fig. 9 (Matsui, Parel, and Norton). Serial stereoscopic angiograms of the same case as in Figure 5.

ported a stereo twin-prism that is placed in front of the objective lens of the fundus camera for glaucoma studies. Stereo fundus photogrammetry has been studied by Crock and Parel¹³ and Crock¹⁴ and Mikuni, Yaoeda, Fujii, and Togano. 15 In 1968 Parel, Crock, and O'Day16 developed an automatic exposure stereoscopic photographic system that has a relatively constant stereoscopic base. Our simultaneous stereoscopic system is much more convenient for stereophotogrammetry because of its absolutely constant stereoscopic base and because both stereoscopic pairs are on the same frame, thus simplifying photogrammetric orientation and reducing processing errors.

SUMMARY

We developed an apparatus for serial, simultaneous stereophotogrammetric fundus angiography by modifying a fundus camera. The paired stereoangiograms obtained by this system provided satisfactory stereoscopy and fair depth of field. With this system, mounting and filing of slides were easy. Accurate photogrammetric analysis of the fundus can be performed. Clinical results were impressive in both young and old subjects with fully dilated pupils.

ACKNOWLEDGEMENT

Mr. Shiro Takizawa, chief of the Optic Technical Division of Topcon Co., Tokyo, Japan, provided technical assistance.

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VIABLE COMPOSITE GRAFTING IN EYELID RECONSTRUCTION

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Composite upper eyelid grafts, which are free full-thickness sections from the upper eyelids, are useful in reconstructing surgical, traumatic, and congenital colobomas of the opposite upper eyelid.1,2 Although the surgeon can often reconstruct large upper evelid colobomas by direct closure after a lateral canthotomy and cantholysis or with a Cutler-Beard³ lower evelid flap, at times there is not enough tissue in the adjacent evelid to allow this.4 In these rare situations, a composite graft must be used. However, because this full-thickness section of the evelid has no blood supply, it must rely on vascularization from the surrounding evelid, and thus has a high failure rate.5,6

The viability of most free grafts is from small buds of vascularized tissue that penetrate them from an overlying or underlying flap. For example, a free skin graft applied over the orbicularis muscle receives its blood supply from buds of blood vessels growing into it from the muscle.

To increase the viability of a composite graft, I devised a new technique that allows vascularization from overlying and underlying flaps. This is accomplished by altering the composite graft to a skin component and a tarsal-conjunctival-margin component. The skin graft is placed against the orbicularis muscle and the tarsal-conjunctival-margin component against a skin flap.

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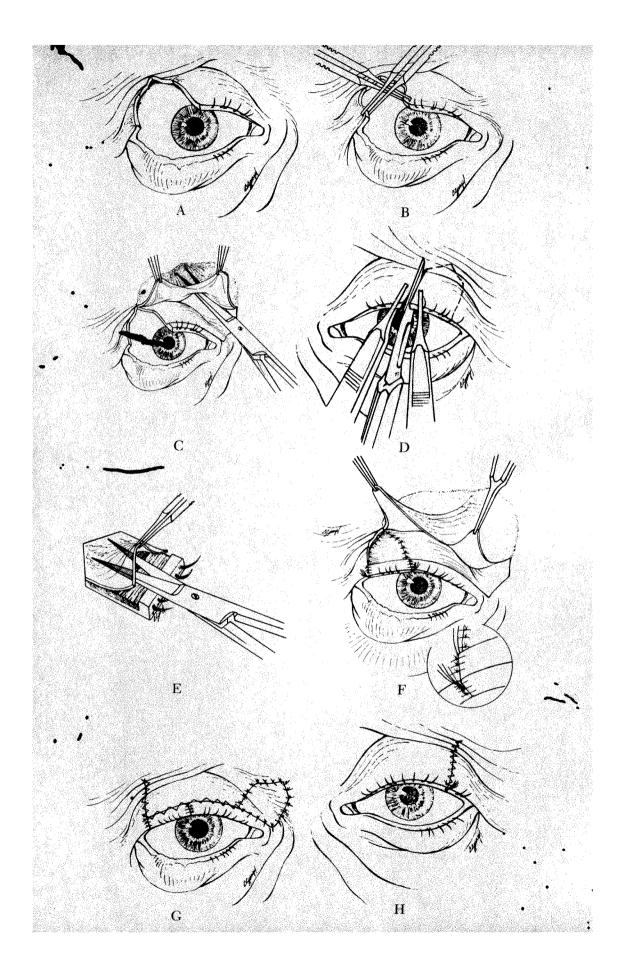
MATERIAL AND METHODS

The surrounding 1-mm edge of the upper eyelid coloboma is excised with a No. 11 Bard-Parker blade to provide a raw edge for the composite graft to rest against (Fig. 1, A). The size of the composite graft, which is circumscribed with a marking pen on the opposite upper evelid, is equivalent to the defect in the colobomatous upper eyelid when the nasal and temporal evend remnants are brought as close as possible to each other with slight tension (Fig. 1, B). The part of the evelid the graft is taken from is usually equivalent to the segment of the affected eyelid. (For example, if the coloboma is in the temporal aspect of the eyelid, the composite graft is taken from the opposite temporal eyelid).

A No. 15 Bard-Parker blade is used to make a skin incision 2 mm above the evelashes over the larger segment of remaining eyelid, beginning at the edge of the coloboma and extending to the opposite extreme of the eyelid segment (Fig. 1, C). An oblique skin incision is then made from the end of the supralash incision, angulated slightly away from the coloboma site. The skin is then undermined from the orbicularis muscle over this eyelid segment with a sharp pointed iris scissors (Fig. 1, C). Observing the points of the scissors beneath the translucent skin facilitates dissection of the skin flap from orbicularis muscle without skin penetration.

The composite full-thickness graft is then taken from the opposite upper eyelid by grasping the eyelid margin on each side of the marked line with two forceps, thus penetrating the eyelid about 5 mm from the margin (Fig. 1, D). A slicing

From the University of Illinois Eye and Ear Infirmary, Chicago, Illinois. Supported by grants from Research to Prevent Blindness, Inc., and the Illinois Society for the Prevention of Blindness.



motion then severs the eyelid margin. This method provides a smooth, square, sharp cut through the margin and aids in approximation of the eyelid later in the procedure. A similar cut is made at the site of the other edge of the composite graft. Westcott scissors are used to create vertical and then oblique cuts from the superior aspect to remove a full-thickness pentagon of the upper eyelid that matches the coloboma.

The composite graft is then divided into two sections (Fig. 1, E). A No. 15 Bard-Parker blade is used to incise the skin 2 mm above the cilia across the graft. The skin is undermined from the orbicularis muscle and placed in saline solution. The orbicularis muscle is then undermined from the levator aponeurosis, adherent to anterior tarsus, and is excised and discarded.

The tarsal-conjunctiva-margin graft is then sutured into the coloboma site (Fig. 1. F). Three 6-0 black silk sutures unite the temporal eyelid margin of the graft to the temporal eyelid margin of the coloboma. One suture passes through the squared corner where tarsus meets conjunctiva, a second suture passes through the gray line, and a third through the first row of eyelashes closest to the gray line (Fig. 1 F, bottom right). These sutures are then tied in triplicate and the two eyelash suture ends are tied over the four internal suture ends, so that when all six ends are severed, they point away from the cornea. The nasal eyelid margin of the graft is then sutured to the nasal coloboma in the same way. Superficial tarsus and levator aponeurosis of the graft are then sutured to superficial tarsus and levator aponeurosis of the eyelids surrounding the graft with interrupted 6-0 polyglycolic (Dexon) sutures. The skin above the eyelid margin of the graft is sutured to surrounding skin with interrupted 6-0 black silk sutures.

The skin flap is then brought over the composite graft and sutured with interrupted 6-0 black silk sutures to the graft and surrounding skin (Figs. 1, F and G). In this way, the flap with its blood supply is now in contact with the graft.

By sliding the skin flap over the composite graft, a skin defect is created at the opposite end of the eyelid. This is covered with the skin graft that was separated previously from the composite graft (Fig. 1, G). Skin graft, which now lies against a vascularized bed of orbicularis muscle, is sutured to surrounding skin with interrupted 6-0 black silk sutures.

If direct closure of the composite graft donor site (Fig. 1, H) is hampered by excessive tension of the opposing flaps, a lateral canthotomy and cantholysis can be performed.⁴ The closure is then made with three 6-0 black silk sutures in the eyelid margin, similarly placed to those used to unite the composite graft to surrounding eyelid margin. Several superficial tarsal and levator aponeurosis 6-0 polyglycolic (Dexon) sutures unite the eyelid above the margin, and the skin and orbicularis muscle are closed with a continuous 6-0 black silk suture.

A light pressure dressing is applied over the reconstructed eyelid; the donor eyelid is left without a dressing. The skin sutures are removed five to six days postoperatively, and the eyelid margin sutures

Fig. 1 (Putterman). A, Temporal coloboma of upper eyelid after excision of 1 mm border. B, Two forceps grasp edges surrounding coloboma of the upper eyelid together to determine size of the needed composite graft. C, Skin flap is raised from orbicularis muscle adjacent to coloboma. D, Full-thickness section of opposite upper eyelid is removed in pentagon-shaped section to provide composite graft for colobomatous upper eyelid. E, Composite graft is divided into tarsal-conjunctival-margin and skin components. F, Tarsal-conjunctival-margin component of composite graft is sutured into upper eyelid defect. Three 6-0 black silk sutures unite each side of the eyelid margin of the composite graft to the eyelid margin nasal and temporal to the graft. Skin flap is rotated over the composite graft. G, Skin flap is sutured over composite graft to provide direct vascularization. Skin graft component from the composite graft is then sutured into resulfant defect created by sliding skin flap. Skin graft is placed against orbicularis muscle for its vascularization. H, Direct closure of donor eyelid.

are removed 11 to 14 days postoperatively.

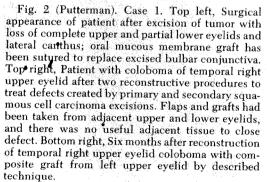
CASE REPORTS

Case 1 A 72-year-old man had a progressively enlarging mass over his right upper eyelid for two years' duration. I first examined him in June 1974 and found an elevated tumor involving the entire right upper eyelid and the temporal one third of the right lower eyelid. A biopsy specimen of this tumor showed squamous cell carcinoma. In July 1974, the entire full-thickness, right upper eyelid was excised from lateral to medial canthus to a level 20 mm above the eyelid margin. The temporal one third of the right lower eyelid and a 2 × 2-cm area temporal to the lateral canthus were also excised (Fig. 2, top left). I performed this surgery under frozen section control until all surrounding edges were free of tumor. The upper eyelid was reconstructed with a tarsal-conjunctival ap from the lower eyelid and sliding skin flaps from the remaining skin of the upper eyelid and temple. The lower eyelid was closed directly to the lateral orbital rim. One week postoperatively, residual tumor was identified on the permanent sections at the temporal edge of the

resected tumor. (This, unfortunately, was missed on frozen section evaluation.) Therefore, two months postoperatively, the tarsal-conjunctival flap was divided and a full-thickness section of upper and lower eyelid on each side of the temporal wound was excised under frozen section control and the resultant colobomas were closed directly. Permanent sections revealed that all the tumor had been removed. The lower eyelid healed satisfactorily, but a wound dehiscence occurred in the upper eyelid, leading to a large coloboma (Fig. 2, top right).

It was impossible to utilize tissue from the lower or upper eyelids to close this defect, because of maximum utilization of these tissues for the original site reconstruction. Therefore, a composite graft from the left upper eyelid was used to reconstruct the upper eyelid defect in December 1976, according to the described technique. A 12-mm horizontal and 15-mm vertical composite graft was placed in the temporal right upper eyelid and a sliding skin flap from the nasal remnant of the right upper eyelid was used to cover it. The skin from the composite graft then filled the resultant nasal defect. The grafts were accepted and the preoperative keratopathy resolved (Fig. 2, bottom right). He has remained without sequelae for 14 months postoperative.











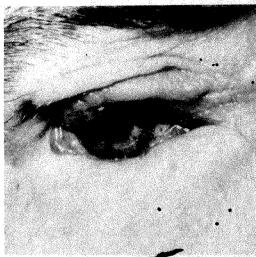


Fig. 3 (Putterman). Case 2. Left, Preoperative appearance of man with right upper eyelid temporal coloboma following industrial accident. Entire right lower eyelid had been reconstructed; it consisted only of mucous membrane graft and skin without any tarsus. Right upper eyelid remnant had also been reconstructed on previous occasions, and no surrounding tissue could be utilized for closing this defect. Right, Four months after reconstructing right upper eyelid coloboma with composite graft from left upper evelid according to described technique.

Case 2-A 42-year-old man was in an industrial accident, which led to the loss of his right upper and lower eyelids. He underwent four surgical procedures before I first examined him in June 1976. He had a coloboma of the temporal half of his right upper eyelid, a lateral symblepharon, and a lower eyelid made up of skin, redundant mucous membrane graft, and scar tissue without tarsus. He also had keratopathy and trichiasis of the remaining upper eyelid; visual acuity was reduced to 6/120 (20/400).

In August 1976, I reconstructed the left lateral cul-de-sac with a buccal mucous membrane graft and performed electrolysis on the misdirected upper eyelid lashes. Postoperatively, the keratopathy worsened and a corneal ulcer developed, which was successfully treated (Fig. 3, left). In December 1976, the coloboma of the temporal upper eyelid was treated with a composite graft from the left upper Lyelid, according to the described technique. The graft measured 15 mm horizontally and 18 mm vertically. The tarsal-conjunctival-margin component was sutured into the temporal eyelid defect and covered by a nasally based skin flap. The skin graft component filled the resultant nasal defect. During the same surgical sitting, the lower eyelid margin was revised by excising scar tissue between skin and mucous membrane and modifying the mucous membrane. Postoperatively, the composite graft was accepted, the appearance of the eyelid improved, and the keratopathy resolved (Fig. 3, right). He has been doing well for four months postoperatively.

SUMMARY

I reconstructed evelids in two patients by successfully using a new technique to increase the viability of a composite, full-thickness evelid graft. Splitting the graft into two components of tarsalconjunctival-margin and skin and placing each component against a viable skin and orbicularis flap, respectively, facilitated the acceptance of the graft and increased the success of the reconstruction.

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SURFACE MORPHOLOGY OF GIANT PAPILLARY CONJUNCTIVITIS IN CONTACT LENS WEARERS

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In a previous report¹ we described a syndrome in contact lens wearers with symptomatology including excess mucus and itching, diminished or destroyed contact lens tolerance, and giant papillae in the upper tarsal conjunctiva resembling vernal conjunctivitis. When the giant papillae form in the conjunctiva, there seems to be an alteration in surface morphology; it would be useful to know how these changes compare with the normal surface. Therefore, we examined the conjunctival surface of subjects with giant papillary conjunctivitis, by scanning electron microscopy.

METHODS

Subjects—We studied the upper tarsal conjunctiva of 11 contact lens wearers with giant papillary conjunctivitis. Five

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wore hard contact lenses and six wore soft contact lenses (Table) made by various manufacturers. Controls consisted of 11 previously reported subjects² with no symptoms and normal conjunctivae, who had never worn contact lenses. Diagnosis of giant papillary conjunctivitis was based on previously established criteria.¹

Biopsies—After local anesthesia, as indicated (Table), biopsy specimens (about 2×2 mm) were shaved off the conjunctiva with a razor blade knife by cutting tangential to the tarsal plate. Biopsy location was restricted to the midcentral upper tarsal conjunctiva. No forceps were

TABLE
BIOPSY SPECIMENS OF UPPER TARSAL
CONJUNCTIVA FROM SUBJECTS WITH GIANT
PAPILLARY CONJUNCTIVITIS

Subject No.	Age (yrs)	Anesthesia Administered*	Contact Lens Typeł
1	19	Nerve block & proparacaine	SCL
2	21	Nerve block and cocaine	SCL S
3	24	Proparacaine	SCL
4	26	Proparacaine	SCL
5	33	Nerve block	HCL
6	33	Nerve block	HCL
7	33	Nerve block	HCL
8	34	Nerve block	HCL
9	53	Nerve block	SCL
10	53	Nerve block	SCL
11	57	Nerve block	HCL

^{*}Van Lint nerve block with 2% xylocaine †SCL indicates soft contact lens; HCL, hard contact lens

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used, nor was the surface of the biopsy specimen touched. Biopsy specimens were slid onto the blade edge during cutting and from the blade edge onto a supporting piece of cardboard, and mounted flat. Mounting the specimen on the cardboard prevented the tissue from folding.

Fixation-Mounted biopsy specimens were immediately immersed in 3% glutaraldehyde solution in 0.15M sodium cacodylate buffer (pH 7.2) at room temperature for three to four hours. Specimens were rinsed in buffer three times and refrigerated overnight. Then they were dehydrated in graded ethyl alcohols, treated with a transitional fluid (Freon 113), and dried in a Bomar SP-900 critical point drying apparatus. Freon 13 was used as a critical point transitional fluid. Specimens were mounted with silver conducting paint on aluminum stubs and coated with thin layers of carbon and gold-palladium in a Hummer sputter coater. Tissues were examined with a scanning electron microscope operated at 20 kV.

RESULTS

Biomicroscopy—Normal upper tarsal conjunctiva may have a satin-smooth, uniform papillary or nonuniform papillary appearance; in giant papillary conjunctivitis, the surface has, by definition, papillae greater than 1.0 mm in diameter. An example of giant papillary conjunctivitis is shown (Fig. 1).

Scanning electron microscopy—At low magnification the general appearance of the surface epithelium (Fig. 1) was markedly different from the normal surface seen in the controls (Fig. 2). Surface cell boundaries were outlined by prominent and altered microvillar borders. Along most borders the microvilli appeared stuck together. These matted microvilli may be an artifact, but specimens from normals did not exhibit this phenome-

non.2 Surface cells were polygonal in shape, but the hexagonal pattern normally seen (Fig. 2) was lost over the top surfaces of papillae; the cells were irregular and frequently elongated (Figs. 1, 3, and 4). Elongation of surface cells was greatest over the central portion of the top of the papillae (Figs. 3 and 4). In the controls, the microvilli gave the surface a uniform shag texture; in the giant papillary conjunctivitis subjects the surface was irregular, although some normal microvilli were present (Fig. 5). In cases of giant papillary conjunctivitis the microvilli had balloon-shaped distal ends, appeared to be adherent (Fig. 5), and formed peculiar white, tufted structures (Figs. 3 and 4). There were two characteristic types of tufted structures. The first type, usually found on the top of papillae, were flattened and the individual microvilli were difficult to distinguish (Fig. 4). The second type, found principally on the sides of papillae, were more elevated and the individual microvilli were easily seen (Fig. 4). There were as few as four to six tufts in some cells and as many as 21 to 25 in others. Cells covering the periphery of the top of the papillae had more tufts than cells on the sides of the papillae (Fig. 4). Cells with more tufts also had a greater surface area (Fig. 4).

Light and dark cells were present in most of the epithelial surface overlying papillae, both in cases of giant papillary conjunctivitis (Figs. 3 and 6) and in normal subjects (Fig. 6). Light cells were more common and were usually smaller and more regularly shaped than dark cells. The more irregularly shaped dark cells had shorter and more densely packed microvilli. Dark cells were similar to those seen in normal subjects,2 slightly depressed from the surface, and had a significantly larger surface area than light cells. More dark cells were on the top of the papillae than on the sides. Occasionally, mound-like elevations were on dark



Fig. 1 (Greiner, Covington, and Allansmith). Top, Everted upper eyelid showing upper tarsal conjunctiva with giant papillae (greater than 1 mm in diameter) and mucus strands (arrows). Eyelid margin is at tip. Bottom, Low magnification scanning electron micrograph of top surface of giant papilla. Note irregularly shaped cells outlined by borders. Both dark and light cells are present. Some have light processes on surface. Sides of papilla are indicated by arrows. Adjacent papilla is present at lower left (bar gauge = 10 µm; ×830),

cell surfaces (Fig. 6). These mounds had raised light centers, were round to ovoid, and were covered with microvilli as in normals. But microvilli from patients with giant papillary conjunctivitis were flattened and matted.

The morphology of small crypts was different from those in the controls (Fig. 7). Although the size of the openings was

approximately the same as in normals (1 to 3 µm in diameter) (Fig. 2),2 the microvillar border pattern showed wide variation in cases of giant papillary conjunctivitis. Variations ranged from almost normal microvillar borders (Fig. 7, top left) to no border at all (Fig. 7, bottom right). Some openings had an unusual surface cell formation around them.



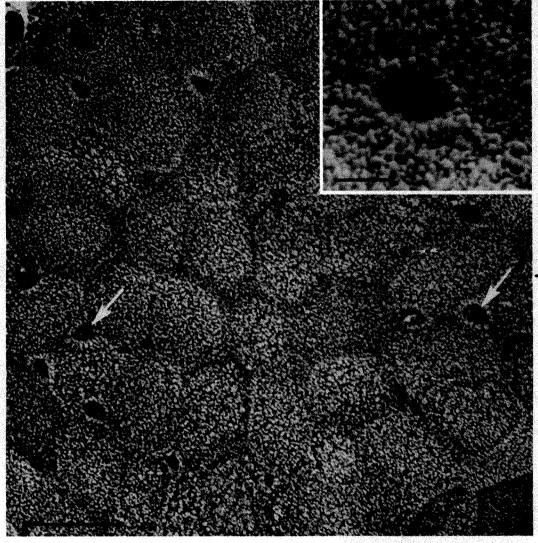


Fig. 2 (Greiner, Covington, and Allansmith). Satin-smooth conjunctival surface and small intercellular crypt openings (arrows) (bar gauge = $10 \ \mu m$; $\times 2,500$). Inset, High magnification of a small crypt opening surrounded by a microvillar border (bar gauge = $2 \ \mu m$; $\times 7,500$).



As in normal surface epithelium,² there were a number of round structures with the same diameter and microvillar border characteristics as small surface crypts, but without any opening (Fig. 5). These round structures were covered with clusters of microvillar-like structures of variable length with balloon-shaped tips.

We observed no crypts of Henle. However, these may have been located between papillae, in valleys we were unable to scan.

DISCUSSION*

The surface morphology of the upper tarsal conjunctiva is dramatically

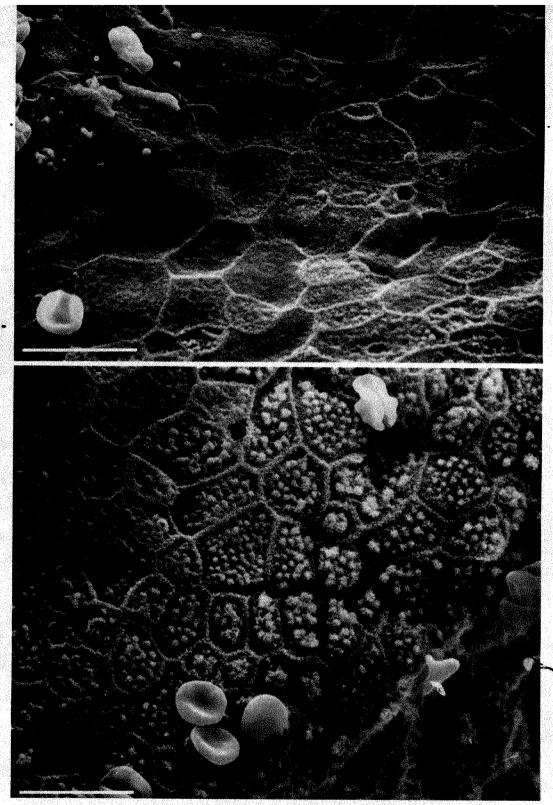


Fig. 3 (Greiner, Covington, and Allansmith). Top, Epithelium of top surface of a giant papilla. Numerous dark cells appear to be depressed from the surface. The small surface holes are 1 to 2 μ m in diameter. There are strands of mucous material at upper left and a red blood cell at lower left. Numerous light cells at lower right have an unusual microvillar pattern. Microvillar borders delineate cell boundaries. Bottom, Side wall of papilla showing cells with white, tufted microvillar structures. Red blood cells can be seen (upper right and lower left). There is a small surface hole (1 μ m in diameter) at top center (bar gauge = 10 μ m; ×3,000).

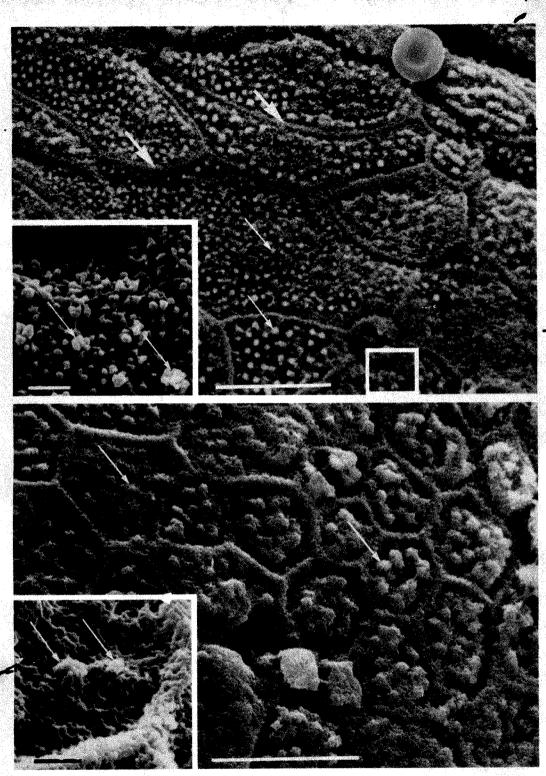


Fig. 4 (Greiner, Covington, and Allansmith). Top, Elongated and irregularly shaped epithelial cells on top surface of giant papilla. Most cell surfaces are covered with microvillar tufts (small arrows). Note prominent cell borders (large arrows). A small surface opening is present at lower right and a red blood cell at upper right (bar gauge = 10 μ m; $\times 3,000$). Inset, Higher magnification of region outlined by square. Some microvilli appear normal, others (arrows) appear stuck to one another, creating a tufted appearance (bar gauge = 1 μ m; $\times 10,800$). Bottom, Side of papilla has smaller and more regular epithelial cells than top surface. A small surface opening is present below center. There are fewer tufts per cell than on top surface (bar gauge = 10 μ m; $\times 3,750$). Inset, Higher magnification of a similar region. Some microvilli are matted, forming a net-like pattern, whereas others appear stuck together to form tufts (bar gauge = 1 μ m; $\times 12,800$).

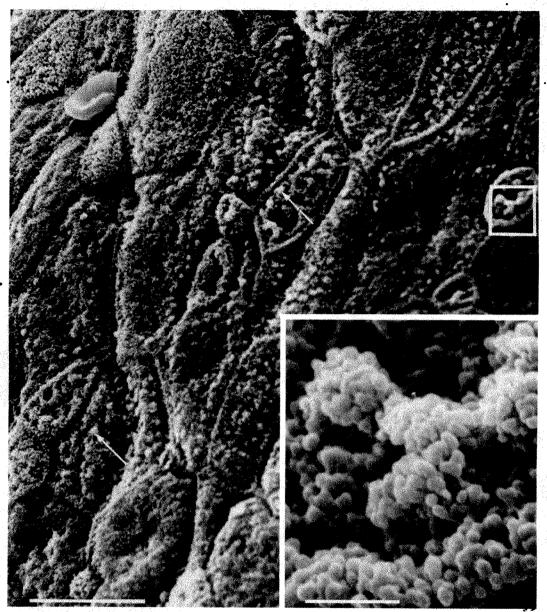


Fig. 5 (Greiner, Covington, and Allansmith). Cells often are outlined by prominent microvillar borders. Round structures (square and below the square to the left) are composed of microvilli with balloon-shaped tips. Arrows indicate microvillar tufts on damaged epithelial cells (bar gauge = $5 \mu m$; ×8,060). Inset, Higher magnification of structure in square showing balloon-shaped tips (bar gauge = $1 \mu m$; ×9,565).

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changed in subjects with giant papillary conjunctivitis as compared to that in normal subjects. We propose several mechanisms of evolution for these surface changes. First, the mushroom-like growth of giant papillae from the deep structures of the conjunctiva might have stretched and distorted the surface; there is at least a twofold increase in the surface area. In

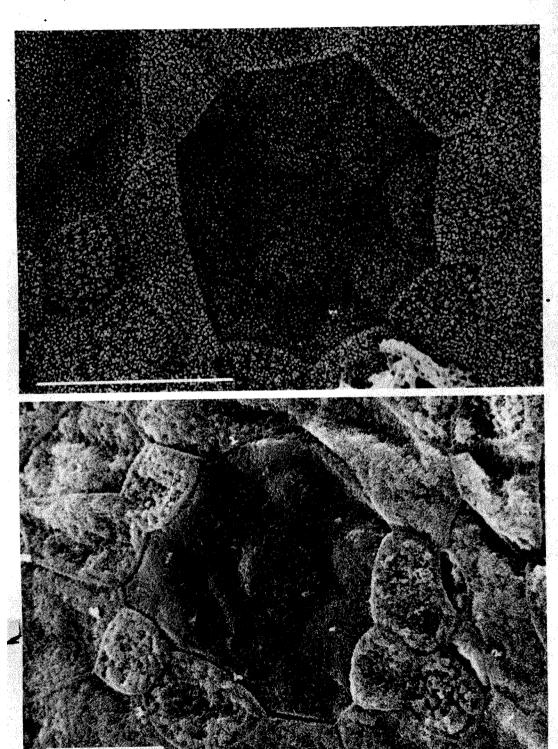


Fig. 6 (Greiner, Covington, and Allansmith). Top, Dark cell surface areas (center and upper left) surrounded by lighter cells with typical surface area and morphology in the normal upper tarsal conjunctiva (bar gauge = $10~\mu m; \times 5,050$). Bottom, Dark cell surrounded by light cells in giant papillary conjunctivitis specimen. Note elevations in the homogenous microvillar field of the dark cells. Light cells have many microvillar tufts. Dark cell appears depressed from the surrounding surface (bar gauge = $10~\mu m; \times 2,800$).

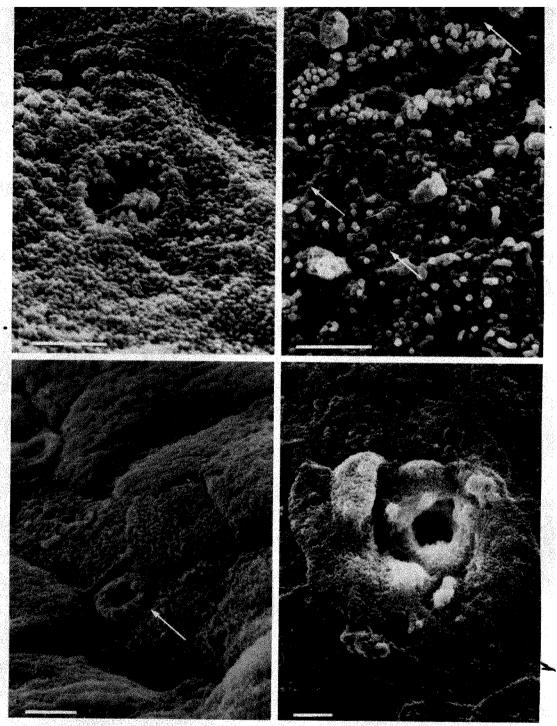


Fig. 7 (Greiner, Covington, and Allansmith). Variability of small surface openings in giant papillary conjunctivitis specimens. Top left, Normal-looking, 1- μ m opening, showing characteristic microvillar border. Surrounding cell surface region is not flat, however, as it is in normals (bar gauge = 1 μ m; ×6,500). Top right, Elongated surface opening is about 2.5 μ m by 1 μ m. Microvilli surrounding the opening differ from normals. Note extensive debris, which is probably mucous. Some is particulate (large particles) and some is strand-like (arrows) (bar gauge = 1 μ m; ×6,500). Lower left, Small 1- μ m surface opening in depression of surface. Border appears smooth, as if the microvilli are flattened and perhaps coated with mucous. Strand-like mucous is present (arrow). Microvilli on adjacent cells are slightly flattened and resemble microplicae. Microvilli on more distant cells are out of focus because the opening is in a valley and, therefore, the other cells are not in the plane of scanning (bar gauge = 1 μ m; ×4,320). Lower right, Small 1- μ m surface opening, with no apparent microvillar border, but surrounded by a flower petal arrangement of cells. Mucous debris is present (bar gauge = 2 μ m ×3,250).

contrast to the normal hexagonal surface pattern, the distorted surface cells of the diseased conjunctiva were elongated and irregular in shape with greater changes on the tops of the papillae than on the sides.

A second possible mechanism for surface changes is mechanical injury. The enormous growth of papillae might bring the upper tarsal conjunctiva into a new and unhealthy relationship to the contact lens or cornea. The upper eyelid, with its extra burden of tissue, blinks and rubs repeatedly over the cornea or contact lens to possibly produce cellular distortions. Cellular damage might even occur as the conjunctiva rests on the contact lens or cornea. Mechanical trauma and injury to the delicate conjunctival surface structures might precipitate changes in cellular metabolism causing the cells to elicit a response altering the cell surface morphology.

The microvillar pattern was altered and the microvillar borders of the crypts were strikingly changed; however, we cannot rule out the possibility that some distortion of the microvillar surface is artifact.

A third mechanism to produce conjunctival surface changes might be the amount, quality, concentration, and distribution of mucus over the surface of the conjunctiva. Excessive mucus is a clinical sign of giant papillary conjunctivitis. Excess mucus may alter some of the surface morphology to produce some of the changes reported, such as microvillar changes.

• A fourth possible mechanism is injury to cell membranes by soluble mediators of inflammation. Among the soluble products expected to be present are the mediators of anaphylaxis released from basophils and mast cells known to be present in abnormal numbers in giant papillary conjunctivitis. Soluble inflammatory mediators of rather low molecular weight, might diffuse through the tissue into the tear film and affect surface morphology.

All of the above mechanisms (stretch, trauma, excess mucus, and mediator injury) are possible sources of change in the conjunctival surface. No evidence to date supports or disproves any of them.

Can the distorted surface of the upper tarsal conjunctiva perform its function adequately? The upper tarsal conjunctiva appears to serve the following purposes: (1) to cover the cornea with a moist surface to prevent desiccation; (2) to lubricate, enabling the evelid to slide easily over the cornea to renew the tear film; (3) to be smooth so that corneal tear resurfacing can be evenly performed; (4) to contribute mucus to the tear film mucus concentration⁵ for whatever functions mucus performs; and (5) to be immunologically competent to react with foreign materials picked up from the corneal tear film. None of these functions appear clinically altered, except for failure to keep excess mucus from blurring the vision.1 Thus, although the distortion at the cell surface level is remarkable, function seems preserved.

SUMMARY

Hard and soft contact lens wearers have been observed to develop giant papillary conjunctivitis in the upper tarsal conjunctiva. We performed scanning electron microscopy on biopsies from 11 contact lens wearers with giant papillary conjunctivitis in the upper tarsal conjunctiva and compared them with biopsies from 11 normals.

Lens wearers had large papillae (>1 mm diameter), which were not seen in normals. We observed dramatic distortion of epithelial cells over central tops of papillae, as well as irregularly shaped cells and loss of the characteristic polygonal shape routinely seen in normals. Epithelial cell boundaries were outlined by prominent altered microvillar borders. Microvilli did not give the surface a uniform shag texture as observed in normals, but were adherent and formed white,

NOTES, CASES, INSTRUMENTS

A NEW HEADREST FOR OPHTHALMIC MICROSURGERY

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Since reporting the development of our earlier prototype¹ for a headrest, we have continued our work and developed a model that meets most of our requirements. This completed instrument retains the elements of complete support and fixation of the patient's head together with hand support for the surgeon, as first described by Dekking.²

This instrument (Fig. 1) is designed to be clamped to an operating table as a projecting extension at the head of the table. It comprises a U-shaped frame of tubular stainless steel with a clamp at

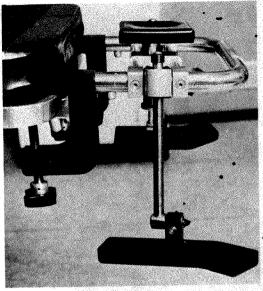


Fig. 2 (Pierse and Steele). The supporting cup for the patient's occiput.

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Reprint request to Arthur D. McG. Steele, F.R.C.S., Moorfields Eye Hospital, City Road, London, England ECIV 2PD each end of the U-limbs. A double-rod cross member halfway along the U-tube carries the head support—a shallow cup of Teflon-coated aluminium, 10-cm square, which supports the occiput (Fig. 2). This cup-plate is mounted on a central

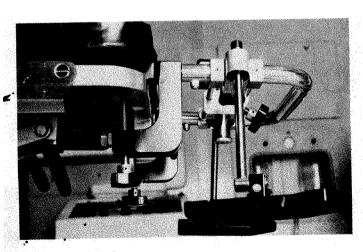


Fig. 1 (Pierse and Steele). The headrest attached to the operating table.

tubular pillar for vertical adjustment. A locking nut then fixes the chosen height.

On each side, between the rod members, a universal joint mounted on the U-frame carries the side arms, which are mounted on vertical rods. The universal joint locking nut then controls the angle of the side arms and their independent heights.

The side arms are made as two horizontal flats, 5 cm wide and 17 cm long; the posterior 5 cm of length being angled inward to the main body at an angle of 30 degrees for better fixation of the temporal region. The patient's head is supported by the 1.5-cm width of the side arms (Fig. 3), also of Teflon-coated aluminum. As the two side arms are totally independent, the entire device is symmetrical. We found it convenient to use the side arm on the same side as the eye for operation, at a lower level than on the opposite side.

We have used this instrument regularly at two-hospitals for over a year. It has been satisfactory for patients having either general or local anesthesia. This

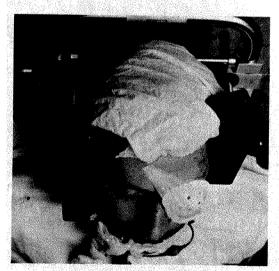


Fig. 3 (Pierse and Steele). The headrest in use.

headrest has been particularly useful for teams engaged in vitreous surgery with the Zeiss Mark VI microscope; the headrest gives excellent control over the visual field and eliminates all extraneous movement.

The following attachments are optional:

Armrests—In addition to hand support, greater dexterity and surgical accuracy can be achieved when the surgeon's forearms also are supported. For surgeons who do not already have this facility in the form of an operating chair, forearm rests have been designed that clamp on to the U-tube. Each rest has independent adjustment for height and angle and provides firm support.

Instrument tray—Most surgeons find it convenient to have a place near at hand to keep frequently used instruments. A detachable tray designed for this purpose fixes to either of the side supporting members after towelling. This tray may therefore be sterilized with the instruments.

SUMMARY

We designed and successfully used a new headrest for ophthalmic microsurgery. The symmetrical instrument has a U-shaped frame and is designed to be clamped to an operating table as a projecting extension. The headrest has been satisfactory for patients having either general or local anesthesia, and particularly useful for vitreous surgery with the Zeiss Mark VI microscope; this headrest gives excellent control over the visual field and eliminates all extraneous movement.

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A SUCTION TIP FOR CONTROLLED REMOVAL OF NONMAGNETIC INTRAOCULAR FOREIGN BODIES

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The two-instrument vitrectomy techniques pioneered by Machemer¹ have facilitated the controlled extraction of nonmagnetic foreign bodies. Often the foreign body can be isolated while still partially suspended in a vitreous scaffold, and the extractor jaws can be easily placed around the object. In this manner, the foreign body is firmly held and the size of the extractor jaws is minimized, thus reducing the size of the exit incision.

On occasion, however, a nonmagnetic foreign body fragment, such as glass, will be encountered lying free on the retinal surface. The jaws of the extractor are then more difficult to position and there is a risk that the jaw tips or the foreign body itself will be pushed against the retina and cause retinal damage, bleeding, or a tear

During the surgical extraction, suction on the vitrectomy instrument is sometimes adequate to pull a small fragment against the metal lips with sufficient force to hold it while the fragment is lifted from the retina and the extractor jaws are used to grasp the fragment. However, leakage from around the opening usually does not allow sufficient grasping power for a larger fragment to be held against the metal opening.

*With the two-instrument vitrectomy technique, I have found it useful to remove the vitrectomy instrument from the fiberoptic light pipe and insert a spe-

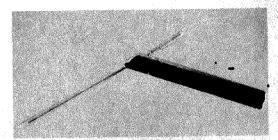


Fig. 1 (Coleman). A 20-gauge needle with plastic sleeve extending approximately 1 mm beyond the metal tip for intraocular suction fixation of non-magnetic foreign bodies.

cial needle encased in a plastic sleeve into the light pipe (Fig. 1).

When suction is applied to the needle, the foreign body is brought against the pliable tip of the tubing and can be held firmly enough to be drawn into midvitreous. In this position, the extractor jaws are easily and accurately applied to the foreign body in the best position for extraction (Fig. 2), so as to minimize the size of the extraction wound.

Extraction forceps as described by Hutton², with silicone covered jaws can be used to hold the fragment. Machinist's tricep forceps (Fig. 3) are

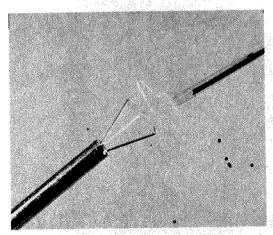


Fig. 2 (Coleman). Once fixation is obtained with the suction device, the foreign body can be optimally positioned for grasping with the extraction forceps.

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also useful for large foreign bodies because of the small size of the wire jaws surrounding the foreign body. This minimizes the size of the extraction incision. Small tricep forceps of this type have recently been described by Wilson.³

In performing the procedure, intraocular infusion can be maintained while the suction tip is inserted through the fiberoptic sleeve by using a syringe attached to the suction tip filled with the same solution as used in the vitrectomy infusion. Infusion is maintained slowly until the suction tip is brought to position immediately adjacent to the fragment. Suction is then applied and the fragment is pulled against the soft pliable opening. Little suction is then required to maintain the fragment as the soft tip is easily occluded by the fragment. The globe does not tend to collapse if the opening is completely sealed. In certain situations, it may be advisable to use a separate infusion needle inserted directly into the pars plana and stabilized by the surgical assistant. Since a large opening is required at the extraction site for the extraction forceps and the foreign body, the assistant may

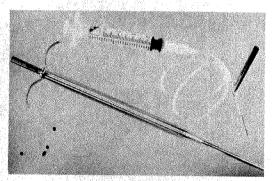


Fig. 3 (Coleman). Tricep forceps for removal of large intraocular foreign bodies. This machinist's tool provides three-point stability with a minimum of added girth required for the exit wound. The forceps are shown with the fully assembled suction device.

place the infusion needle into the same opening used for the extraction forceps.

This instrument technique has been used successfully in lifting fragments away from the retina so that they can be readily removed from the eye with lessened risk to the retina.

SUMMARY

I used a soft-tip suction needle during a vitrectomy procedure to lift glass foreign bodies away from the retina so they could be more easily and safely grasped with foreign-body extraction forceps. The use of the instrument reduces the chance of inadvertent scraping of glass fragments or instruments against the retina during the removal.

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A MAGNET TIP FOR CONTROLLED REMOVAL OF MAGNETIC FOREIGN BODIES

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The removal of ferrous intraocular foreign bodies with a suitable magnet is an accepted method of surgical extraction. To avoid prolonging surgery, the strong-

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est pulling power is desirable. The shortest magnet tip affords the greatest power as the force of the magnetic field decreases with distance from the magnet coil.

The magnet tips are generally designed to be as short as possible to provide maximum power. Too short a length, however, may limit accessibility to the foreign body in the magnetic field. Thus, several shapes of tips have been designed to provide the best compromise between accessibility and power. We generally use the Bronson-Magnion pulsed magnet.* The two tips supplied with the magnet are shown (Fig. 1, left).

In the removal of intraocular foreign bodies, conventional surgical techniques require a scleral incision, and usually, incision of the choroid through the pars plana. Depending on the position of the extraction site, the retina may also be incised. One problem with the subsequent removal of the foreign body is that the lips of the choroidal incision seldom separate well and the foreign body often incarcerates the choroid (and the retina, if present), between the foreign body and an externally applied magnet tip, thus dragging the tissue into the scleral wound as the foreign body is attracted to the magnet. This can produce tearing of the choroid, excess vitreous loss, bleeding, and other complications.

A fine, pointed magnet tip designed to be introduced directly into the eye allows the foreign body to stabilize on the tip of the magnet before extraction, thus permitting a more controlled maneuvering of the foreign body out of the eye. In a magnetic field, magnetic foreign bodies align automatically along their long axes; thus it is usually possible to maneuver foreign bodies so that an edge or tip is presented through the wound, thus minimizing the

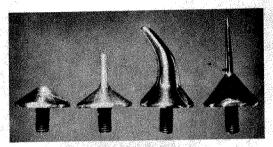


Fig. 1 (Coleman). Magnet tips for use with the Bronson-Magnion pulsed magnet. The two tips on the left are supplied with the instrument. The two tips on the right allow for direct access to foreign bodies, as well as intraocular access (far right).

surface exposure and the resultant retinal and choroidal dragging or even incarceration between the foreign body and the magnet tip. It also reduces the chance of dragging a foreign body across the retina, thus reducing the chances for retinal tear. In this manner, wound complications are minimized.

The only significant disadvantage of the intraocular tip I have designed (Fig. 1, far right) is that the power at the end of the intraocular tip is considerably less than the shorter external tips supplied with the Bronson-Magnion magnet. This disadvantage is offset by the ability to insert the longer intraocular tip closer to the foreign body.

I compared the field strengths of the two tips recommended by Bronson with the intraocular tip (and a curved tip similar to that supplied with the Storz-Atlas magnet, that is useful for posterior orbital foreign bodies) (Fig. 2).

The short tips supplied with the instrument are clearly superior to the other designs in terms of field strengths at the tip. However, when compensation for distance from the foreign body is made to allow for closer approximation of the tip to the foreign body, the differences are insignificant. Adequate magnet strength usually remains with the intraocular tip to perform the desired function. When suffi-

^{*} Available from Storz Instrument Company, St. Louis, Missouri.

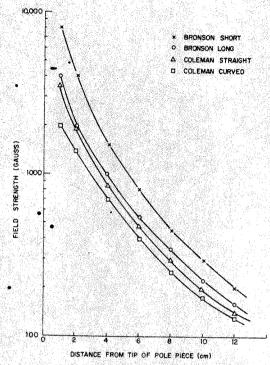


Fig. 2 (Coleman). Field strengths for the four magnet tips demonstrate that the longer, specialized intraocular tip has less pulling power, but its small distance from a foreign body usually compensates to provide adequate strength.

cient strength is not present, the shorter tips may be easily substituted.

SUMMARY

I designed a small diameter magnet tip for intraocular removal of foreign bodies, particularly at the time of vitrectomy. Advantages of the intraocular tip include: (1) reduced likelihood of scraping of the foreign body along the retinal surface while in the magnetic field; (2) reduced chance of incarcerating the retina or choroid, or both, between the magnet and the foreign body during removal. Although the intraocular tip is less strong than conventional shorter tips, it provides greater control in the removal of intraocu-

lar foreign bodies when sufficient magnet strength is present.

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SCLERAL RING AS TEMPLATE FOR CORNEOSCLERAL GRAFT

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A scleral ring provides an easily availae template, in variable sizes, to outline a

ble template, in variable sizes, to outline a large corneoscleral graft in cases of anterior segment ectasia or staphyloma.

CASE REPORT

A 19-year-old-woman sustained a corneal laceration in her right eye while throwing rocks at a bottle on Jan. 24, 1976. After 48 hours of topical antibiotic therapy in the hospital, she developed a purulent corneal ulcer that subsequently grew alpha hemolytic streptococcus. With hourly topical and systemic antibiotics, the superior comea thinned to half thickness; 17 days after the injury a conjunctival flap procedure was performed. One month later the lens had opacified and the cornea began to bulge forward beneath the conjunctival flap so that the eyelids could not close. Visual acuity without correction was R.E.: light perception, and L.E.: 6/6 (20/20). The left eye was normal. In the right eye the area of ectasia encompassed the superior three fourths of the cornea and extended 1 mm into the sclera. Uveal tissue adhered to the posterior surface of the vascularized cornea. The intraocular pressure was high by palpation. Contact B-scan ultrasonogra-

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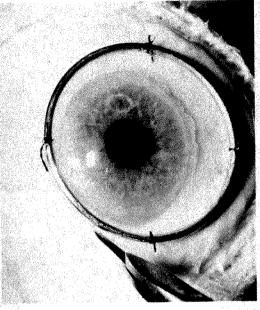


Fig. 1 (Waring and Beernink). Scleral ring sutured to donor eye forms 15-mm diameter template for razor blade knife.

phy showed echoes within the anterior staphyloma, a lens in normal position, and an accoustically clear vitreous.

On Aug. 15, 1976, we performed a limbal conjunctival peritomy and sutured a 23-mm single scleral ring to the sclera. The base of the ectatic cornea measured 15 mm with calipers. We sutured a 15-mm single scleral ring to the intact donor eye in a slightly eccentric position and cut a partial thickness groove around its outer edge (Fig. 1). After removing the ring, we entered the anterior chamber with a razor blade knife and excised the donor corneoscleral button with corneal scissors. We sutured the same 15-mm scleral ring to the base of the host ectasia, outlined the incision with a razor blade knife (Fig. 2), removed the ring, entered the globe, and excised the ectatic sclera and cornea. Since the ectasia was eccentric, removal of the scleral portion left a cyclodialysis superiorly of approximately four clock hours. After a total iridectomy, planned extracapsular eataract extraction (the anterior lens capsule was already broken), and anterior vitrectomy with a vitrectomy instrument, we sutured the corneoscleral graft in place with interrupted 10-0 nylon and 6-0 mersiline sutures.

Three months postoperatively, the intraocular pressure by Mackay-Marg tonometry was 18 mm Hg, the graft was 0.62 mm thick centrally, and the macula and optic disk appeared normal by indirect

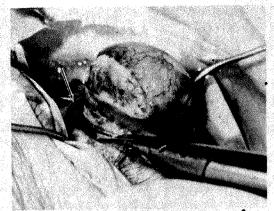


Fig. 2 (Waring and Beernink). Scleral ring (arrow) sutured at base ectatic cornea forms 15-mm diameter template for razor blade knife.

ophthalmoscopy. Over the next six months, we successfully treated a gram-positive bacterial corneal ulcer with topical and subconjunctival gentamicin and methicillin, a nonhealing epithelical defect with a soft contact lens, an immunologic endothelial graft rejection with topical and subconjunctival prednisolone acetate, and a secondary increase of intraocular pressure with one 270-degree cyclocryotherapy. One year postoperatively, the patient manifested a retrograft fibrous membrane with mild corneal edema, a visual acuity of 6/60 (20/200) with correction and an intraocular pressure of 16 mm Hg by Mackay-Marg tonometry.

DISCUSSION

The ophthalmic surgeon who performs corneal grafts larger than 12 mm in diameter, the size of the largest commercially available trephine, must match the size and shape of donor and host tissues by a method other than a trephine. He may excise the host tissue freehand, then cut the donor tissue larger than needed, suture it in one area of the host, and directly fashion the remainder with knife or scissors to match the host defect. If the host cornea is flat, he may use a template of paper, plastic, wax, or malleable metal as an outline. Alternatively, he may modify a drafting compass by blunting the central arm to avoid penetrating the globe; the movable compass arm can then be tipped by a marking pencil or razor blade to create an accurate circle.

When the cornea is ectatic, all these methods are more difficult. A single scleral ring provides a readily available and variably sized template which insures a uniform circular outline when sutured to both donor and host globes. The knife blade does not actually hug the edge of the ring, as this displaces it slightly and might cut the fixation sutures, but gently follows the outline of the ring while making a partial thickness groove.

SUMMARY

A 19-year-old woman sustained a corneal laceration in her right eye. After bacterial keratitis was treated by antibiotics and a conjunctival flap, she developed a corneoscleral ectasia. We used a 15-mm diameter scleral ring as a template for corneoscleral graft. We obtained uniform donor and host buttons by suturing the ring alternately to donor and host eyes. One year postoperatively, the patient had a retrograft fibrous membrane with corneal edema, a visual acuity of R.E.: 6/60 (20/200), and intraocular pressure of 16 mm Hg.

OPHTHALMIC MINIATURE

He was harnessed in a pair of spectacles, so admirably fitted to the prominences and depressions of the orbitary processes, that one might have taken the whole optical apparatus for the natural production of the parts, or an expansion of the cornea spread out upon a delicate frame of silver wire.

The Lancet, March 12, 1825

MEETINGS, CONFERENCES, SYMPOSIA EDITED BY THOMAS CHALKLEY, M.D.

THE OCULAR MICROBIOLOGY AND IMMUNOLOGY GROUP MEETING

The 1977 meeting of the Ocular Microbiology and Immunology Group was held at the Sheraton Dallas Hotel in Dallas, Texas, Oct. 1, immediately preceding the meeting of the Academy. Dan B. Jones, executive secretary, opened the proceedings and then turned the program over to the moderators, Peter Liabson for the morning session and Jeffrey Lanier for the afternoon session.

The first several papers dealt with viral infections.

M. P. Langford, G. J. Stanton, J. C. Barber, and S. Baron reported an accidental human infection by coxsackievirus type A24 (CA 24) in a young technician. The disease ran the same course as that reported for this epidemic picornavirus conjunctivitis, and CA 24 was isolated from tear samples. Although epidemic picornavirus conjunctivitis and acute hemorrhagic conjunctivitis, both highly contagious eye infections, have caused epidemics in many areas of the world, no American reports have been published. In the case reported here, there was neutralizing antibody activity in the tears within 24 to 48 hours.

In vitro inhibition of adenovirus replication by trifluorothymidine was reported by R. A. Eiferman and D. A. Lennette. Several discussants pointed out that since the levels attained in vitro cannot be attained in vivo, this drug is not clinically helpful.

G. Mintsioulis, C. Dawson, and J. O. Oh reported on herpes simplex virus infection of the eye after infection of the superior cervical ganglion. Persistently infected sensory neurons are the principal

reservoir of herpes simplex virus between disease episodes. In this study the superior cervical ganglion was infected to see what type of ocular disease would result. Among the virus-positive rabbits, conjunctivitis or keratitis developed in 61% of the animals and iritis in 94%. The corneas of rabbits (but not of man) have a rich adrenergic supply, which no doubt accounted for the high incidence of keratitis. In man, infections of the superior cervical ganglion are probably associated primarily with bouts of iritis.

Y. M. Centifanto, Z. S. Zam, and H. E. Kaufman tested the cell-mediated immune response of patients with recurrent herpes simplex virus infections by a direct assay of leukocyte migration inhibition factor. It appears that patients with recurrent disease have depressed cell-mediated immune responses between and during attacks of active disease.

G. Smolin, M. Friedlaender, and M. Okumoto presented two papers on the efficacy of levamisole in herpetic infections in rabbits. Levamisole is an immunomodulator that can enhance cell-mediated immune responses in depressed hosts. The authors discussed their model for the production of chronic herpetic keratitis, which they achieve by means of corticosteroids. Using this model, they found that levamisole was effective in preventing stromal disease and in speeding up the healing of the milder epithelial lesions. In their second experiment, the left eyes of these rabbits were infected with herpes simplex virus after the initial infection in the right eyes had healed. The levamisole-treated group had milder clinical disease in the second eye than the untreated group. Viral assays showed lower titers in both of the levamisoletreated groups.

T. O. Wood and R. Deshaies presented data showing that trifluorothymidine is effective against ulcers resistant to idoxuridine and adenine arabinoside, and against dendritic ulcers that appear while patients are using idoxuridine. Side effects were minimal, but it was noted in the discussion that they are occuring more frequently, now that use of the drug is increasing.

C. M. Kalsow and W. B. Wacker described an experimental allergic uveitis
that was induced in guinea pigs with extracts, of homologous pineal gland homogenates.

M. Allansmith and J. V. Greiner reported that normal conjunctivae can contain nearly as many inflammatory cells as the maximum number found in infected conjunctivae, and that there must be chemotactic factors for neutrophils and lymphocytes in the tear film to account for the presence of these cells in the epithelium.

The normal rat conjunctival epithelium contains peroxidase, a microbiocidal enzyme. The next group of investigators (R. Malaty, B. Nichols, and C. Dawson) were interested in learning whether the peroxidase plays a role in eliminating *Chlamyddia* during the resolution of chlamydial infection in guinea pigs. The results of the experiment suggested (1) that peroxidase is activated by both infective agents and noninfectious foreign material, and (2) that peroxidase does not participate in the elimination of the infectious agent since it was not detected *Chlamydia*-containing vacuoles.

H. Gelender and R. K. Forster presented data on the use of cytology in diagnosing extraocular tumors. Papsmear cytology in their study proved to be useful in evaluating suspicious benign and neoplastic extraocular lesions. Although the authors concluded that cytology was a valuable diagnostic procedure, some discussants believed that biopsy

should be performed in all suspicious cases regardless of the outcome of the cytologic examination. It was also mentioned that Giemsa-staining was easier and the results comparable, and that if a specimen was sent to a general pathologist, it should be ascertained whether or not he was familiar with ophthalmic histology.

An interesting report on ligneous conjunctivitis was presented by D. Swanson and F. Meronk. Biopsy of the lesion showed many lymphoid follicles and a marked increase in the concentration of mast cells. Treatment with cryotherapy, topical dexamethasone, and disodium cromoglycate seemed to be effective. One discussant offered substantiating evidence that this disease had an immunologic basis by noting that there were eosinophils in a case of his and that treatment was successful with disodium cromoglycate alone.

The morning session closed with the report of S. A. Fowler, H. I. Covington, J. V. Greiner, and M. Allansmith on a scanning electron microscopic study of contact-lens-associated giant papillary conjunctivitis. A few wearers of hard and soft contact lenses develop giant papillary conjunctivitis in the upper tarsal conjunctiva. This conjunctival response does not damage the cornea but takes many months to clear after lens removal.

The afternoon program began with the report of M. Blumenkranz and J. P. McCulley on a heroin addict who was referred for treatment of a recurrent uveitis. The patient had been treated for endophthalmitis six weeks previously. Repeated vitreous aspirations failed to yield microorganisms, but the eye became blind and painful and was enucleated. Intraocular infection with Candida was then demonstrable. The authors believed clinical appearance was perhaps the most reliable guide to the diagnosis of Candida

endophthalmitis in the drug abuser, especially since vitreous cultures might not be helpful early in the course of the disease. Several discussants suggested that it was important to search for systemic lesions as well.

C. S. Foster and E. J. Goetzl presented a case of impaired neutrophil and monocyte chemotaxis in a patient with atopy, hyperimmunoglobulinemia E, and recurrent infection. This patient received ascorbate therapy and showed increased monocyte chemotaxis. During the discussion it was mentioned that levamisole had been effective in a similar case.

D. B. Jones and J. A. Smelley presented a case of nematode keratitis. The actively moving nematode was probably the third-stage larva of Ancyclostoma caninum. As was anticipated in this probably systemic disease, treatment with thiabendazole was effective. The clinical course was interesting because the gyrating worm disappeared, leaving a focal episcleritis and multiple pleomorphic stromal infiltrates that slowly cleared over an 18-month period. Topical corticosteroid therapy was used to accelerate the clearing.

In a study conducted by J. H. McNatt and L. A. Wilson on the anaerobic flora of the normal conjunctival sac, obligate anaerobes were recovered from 52% of the eyes cultured. *Propionibacterium acnes*, the most frequently encountered anaerobe, was present in 50% of the eyes. Aerobic or facultative anaerobic bacteria were present in 33% of the total eye cultures, with *Staphylococcus epidermidis* the organism most commonly recovered. It is possible that the origin of most of the bacteria in the eye is the skin of the eyelids.

D. O'Day and J. H. Elliott described a corticosteroid-antibiotic regimen for the management of keratitis caused by infection with *Pseudomonas aeruginosa*. All of

the patients were treated by appropriate, specific, local and systemic antibiotic therapy, based initially on anticipated sensitivities but modified when indicated. Local corticosteroid was introduced one to 14 days after antibiotic therapy was begun, once clinical improvement was noted or sensitivity studies showed that the appropriate antibiotic was being used. The antibiotics usually used were gentamicin, colymicin, or carbenicillin. The authors believed this therapeutic regimen compared favorably with any other that has been described. One discussant emphasized the emergence of resistant strains of Pseudomonas and the dangers of corticosteroid therapy in microbial infections, especially Pseudomonas infections. H was suggested that for comparative purposes the authors treat a comparable group without adding a corticosteroid.

According to R. W. Wolters, J. Jorgensen, and R. H. Poirier rapid detection of minute amounts of gram-negative endotoxins is an acceptable method for the early diagnosis of gram-negative infections. Limulus assays were performed 24 hours after the induction of gramnegative corneal infections in rabbits. The assay was reliable in the authors' hands, but one discussant noted that ubiquitous contaminants can result in false-positive results.

The data presented by F. P. Furgiuele, N. Cameron, J. Sassani, and D. Cameron showed that tobramycin, if given early enough by the subconjunctival route, was an effective treatment for experimentally induced staphylococcal endophthalmitis. Intravitreal injection at the beginning of therapy was no more effective, but if subconjunctival hydrocortisone was added, there was marked improvement in the course of the infection. Tobramycin and gentamicin appear to be equally effective in this model.

A case of Aeromonas hydrophila corne-

al ulcer was presented by F. T. Feaster, R. M. Nisbeth, and J. C. Barber. The course of the disease and its resolution by treatment with topical and systemic gentamicin were described.

S. Stenson, R. Newman, P. Freyne, and S. Rosenthal presented data indicating the presence of microorganisms in McCarey-Kaufman-stored keratoplastv donor material despite broad-spectrum antibiotic coverage (penicillin-streptomycin). Staphylococcus aureus (coagulasepositive), was present in 3% of the solutions examined, and Rhototorula and cepacia in 1% each. It was suggested during the discussion that these figures might actually be too low since the results of oulture attempts in the presence of an antibiotic are probably inaccurate. It was also noted that gentamicin is now usually used instead of the penicillin-streptomyein combination.

According to N. Keller, L. Campbell, and J. Anderson, experimental cataracts were induced, in vivo by the intravitreal injection of 1.0 or 10.0 µg of benzalkonium chloride.

M. Barza, A. Kane, and J. Baum reported the levels of gentamicin in the ocular tissues of rabbits that had received sub-

conjunctival injections of the drug. The concentration of gentamicin in most of the ocular tissues of normal eyes was higher than in the tissues of inflamed eyes, the drug probably having been lost through the dilated blood vessels of the inflamed eyes. Retrobulbar injections in rabbits resulted in orbital drug loss, lower doses reaching the posterior portion of the globe by this route than by subconjunctival injection.

According to R. Abel, G. L. Boyle, and S. M. Podos, intraocular penetration of amoxicillin in humans was comparable to the levels attained with ampicillin. These orally administered antibiotics apparently cannot be maintained in theaqueous humor at therapeutic levels that would be necessary to reverse an established intraocular infection.

Dan B. Jones continues as executive secretary. His excellent leadership was affirmed and appreciated. It was also agreed that an abstract of each paper is published in the Transactions of the Academy of Ophthalmology and Otolaryngology.

GILBERT SMOLIN

AMERICAN JOURNAL OF OPHTHALMOLOGY

FRANK W. NEWELL, Editor-in-Chief 233 East Ontario St., Chicago, Illinois 60611

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JONATHAN HUTCHINSON—1828-1913

Can you name the dermatologist who became president of the Ophthalmologic Society of the United Kingdom? The ophthalmologist who was named president of the 1896 International Dermatology Congress? The generalist who was elected to the presidency of the Pathological Society of London? The syphilologist elevated to the presidency of the Royal College of Surgeons? The surgeon elected as president of the Neurological Society?, and so forth. This is the 150th anniversary of the birth of the man who filled all of these roles and filled them with dignity and distinction. An individual who was one of the most remarkable products of Victorian England and one of the greatest physicians of all time, Sir Jonathan Hutchinson.

Hutchinson was perhaps the greatest "generalist" in medicine. He was opposed to specialization and stated1:

Our existing specialists are chiefly founded on groupings: (1) According to the organ or part affected, and this is arbitrary and unnatural or, (2) according to cause, and this presupposes diagnosis. The human body is one whole. Year by year the specialist loses hold of the general knowledge he acquired in early life, and his range of investigation becomes narrower.

Notwithstanding his antipathy for specialists, he was so astute in pursuing his special interests that he was widely recognized both by his peers and the lay public as a great neurologist, dermatologist, internist, general surgeon, and ophthalmologist. He provided the first clinical descriptions of more medical conditions, and more clinical signs of importance than any physician before his time or since. His medical work was rewarded with virtually every honor England could offer; honorary degrees from every major university, Fellowship in the Royal Society, presidency of every medical group in which he took an interest, and, of course, knighthood.

Had he contributed nothing to clinical medicine Hutchinson would still be justly regarded as an important figure in medicine. He was an indefatigable editor

and writer. Not only did he serve for a short time as a forward-looking editor of the British Medical Journal, but he was the founder and major force behind the New Sydenham Society, and during the years he served as its secretary (1859-1907) it issued 190 volumes of the best in medical literature. It was this society that commissioned, translated, and published Donders classical treatise, "On Anomalies of Accomodation and Refraction of the Eye." Of course, he will always be remembered for his Archives of Surgery, which he founded in 1889 and for which he was the sole contributor for the 11 years of its existence. Of these volumes, which are mainly clinical descriptions, Osler² wrote:

When anything turns up which is anomalous or peculiar, anything upon which the text-books are silent and the systems and cyclopedias are dumb, I tell my students to turn to the volumes of Mr. Hutchinson's Archives of Surgery as, if it is not mentioned in them, it surely is something very much out of the common.

Had he no clinical practice and had he done little writing he would still be counted among the greats of medicine. He was by all accounts a brilliant lecturer and organizor of medical facts. Abraham, writing many years later, recalls:

I can still remember the first time I saw Jonathan Hutchinson. It was at the Polyclinic in Chenies Street, and he was addressing a gathering of medical men. He was then almost a legendary figure to most of us. . . I do not remember what he talked about that day—but he held us completely for an hour. He spoke rather slowly and solemnly, and what he said was clear and logical. There was nothing scintillating about it, but you felt he was speaking from immense knowledge.

He established postgraduate teaching institutions for he firmly believed in continuous education throughout one's professional life. "He had wanted to make it, and London, a great postgraduate centre; but he was then, and even now forty years later, much before his time." ³

Had he not entered medicine at all Jonathan Hutchinson would undoubted-

ly be numbered as one of the remarkable individuals of the past century. He personally founded a number of natural history museums. The one at Haslemere, in the south of England, still flourishes. These museums were and are outstanding examples of living institutions that provide a rich learning experience for all who enter them, regardless of age or intellect: "My wish is that even uneducated persons should be enabled to grasp the great principles of Biology. I shall endeavor to make elementary and general knowledge an easy acquisition to all."3 In his museums, Hutchinson instituted a policy of asking those who attended to take an active role and to answer questions about what they had seen and heard. He was well aware of the value of "programmed" text material.

While Hutchinson was not a genius in the sense of having a super intellect, he was highly intelligent and he combined intelligence with tremendous inner drive. His mission in life was to disseminate knowledge and dispel ignorance. His contributions to clinical ophthalmology are vast and oftentimes unappreciated by the modern generation of ophthalmologists who tend to be enamored by new techniques, new operations, new medications, and who often forget they have a past worth remembering and worth cherishing. Some of Hutchinson's contributions will appear as ophthalmic miniatures in subsequent issues of THE JOUR-NAL during this, his anniversary year.

PAUL HENKIND

REFERENCES

1. Hutchinson, J.: quoted by Hutchinson, H.: Jonathan Hutchinson: Life and Letters. London, Wm. Heinemann. 1946, p. 84.

2. Osler, W.: The importance of post-graduate study. Lancet 2:73, 1900.

3. Abraham, J. J.: Introduction to Jonathan Hutchinson: Life and Letters. London, Wm. Heinemann, 1946, pp. 1; 174.

INTERPROFESSIONAL **EDUCATION IN OPHTHALMOLOGY**

Organized ophthalmology, like other medical specialties, has traditionally confined its educational efforts to its own members. This approach has ignored a need, or assumed there is none, to teach other health professionals about ophthal-

mology.

Recently, however, the American Academy of Ophthalmology, a division of the American Academy of Ophthalmology and Otolaryngology, has modified this approach. The Continuing Education Secretariat through its Interprofessional Education Committee and the Academy's Public and Professional Education Committee have begun programs to teach ophthalmology to health professionals beyond the Academy's ranks; programs that eventually will include lectures, workshops, and exhibits at national meetings, articles in professional journals, and selfinstructional materials.

Initially, however, it seemed appropriate to provide materials for ophthalmologists to use themselves when teaching about ophthalmology at the local level. Accordingly, a slide-script program has been produced and is now available for purchase from the Academy. Entitled "Introduction to Ophthalmology," the program contains 52 slides and a suggested script. With this material, ophthalmologists have access to professionally prepared slides and can use them in any way they wish. For those wanting a "canned" lecture, the script organizes the slides into a 30-minute talk that can be presented to nurses, allied health groups, or laymen.

Further plans include color illustrated booklets of the slides and script for ophthalmologists to use in their offices and audiocasettes for those who wish a slide-tape program. Other programs are planned to help teach other medical specialists about the eye, such as one oriented toward emergency physicians.

These efforts by the Academy emphasize ophthalmologists' interest in providing educational materials for physicians and other health care personnel outside of ophthalmology. This venture may lead to cooperative efforts whereby ophthalmologists may learn more about other areas of medicine from other specialty societies.

PAUL R. LICHTER

OBITUARY

John H. Dunnington, M.D. 1894-1977

Colleagues, patients, and friends were saddened to learn of the death of John Hughes Dunnington on Oct. 17, 1977.

Born Jan. 12, 1894, he was the son of India Wyckliffe Knight and Walter Grey Dunnington, Jack Dunnington's father wanted all his sons to enter college together. So Jack, the youngest, enrolled at Hampton-Sidney College at 14 years of age. In later years he was to say that at 14 he was too young to enjoy the intellectual and social pleasures of college life but he was an outstanding student. He was a loyal alumnus and in 1952 Hampton-Sidney awarded him an honorary doctorate of law degree.

After receiving a medical degree from the University of Virginia in 1915 at the age of 21, he took his residency training in ophthalmology at Manhattan Eye, Ear and Throat Hospital and then entered the Army Medical Corps. Here he met John M. Wheeler who was to have a vital influence on his career. After World War I, Dr. Dunnington entered private practice in New York City. At the same time he worked and taught at New York Eye and Ear Infirmary, Bellevue Hospital,

and the University and Bellevue Medical College. When the Institute of Ophthalmology was founded in 1929 under the aegis of Dr. J. Wheeler, Dr. Dunnington accompanied him and thereafter was associated with the Columbia-Presbyterian Medical Center and the College of Physicians and Surgeons of Columbia University. In 1940 he became professor of ophthalmology and, on Dr. Wheeler's death in 1942, acting director of the Institute of Ophthalmology. In 1944 he was appointed director, a post he held until 1959. Throughout this period, he developed a department of academic pre-eminence dedicated to the highest caliber of clinical ophthalmology. The numerous Dunnington residents were measured by his own exacting standards, and his astute judgment was demonstrated at his rounds, which became classic. After retiring as director of the Institute in 1959, Dr. Dunnington continued to practice aphthalmology until his death.

Dr. Dunnington's career spanned an epoch during which enormous advances were made in the scientific aspects of ophthalmology. He encouraged basic and clinical research. He was both intensely curious and charmingly critical about experimental designs and was often impatient to see the result. At the same time, he was primarily the clinician stressing the importance of careful examinations, meticulous surgical technique, and attentive patient care. These were emphasized in his enormously popular Academy courses on strabismus and cataract surgery. Through them he influenced generations of American ophthalmologists. For much of his career Dr. Dunnington was particularly interested in motility problems, and published numerous articles on the various aspects of strabismus between 1920 and 1950. His thesis for the American Ophthalmological Society was, "Tenotomy of the inferior oblique." Later in his

career, his interests shifted to cataract surgery and particularly to surgical wound healing.

Dr. Dunnington ably served ophthalmology in many areas. He was a frequent examiner of the American Board of Ophthalmology and was chairman from 1950 to 1952. He was a member and past president of the American Ophthalmological Society and the American Academy of Ophthalmology and Otolaryngology, a member of the American Board of Plastic Surgery, American College of Surgeons, and the Canadian Ophthalmological Society. He was a member of the Editorial Board of the Archives of Ophthalmology for many years, a director of Recording for the Blind, ophthalmic advisor for the Knights Templar Eye Foundation, Inc., and honorary vice-president of the National Society for the Prevention of Blindness. He received the Howe Medal in 1954, was an honorary member of the Ophthalmological Society of the United Kingdom, the Ophthalmological Society of Northern Greece, and the Pan American Association of Ophthalmology. His honoray lectures included the Schönberg and Gifford in 1950, the Jackson Memorial, 1951; Bedell, 1953; Bowman, 1955; Proctor, 1956; and deSchweinitz, 1957.

Dr. Dunnington often said that his hobby was sitting and perhaps it wasduring his summer holidays on Nantucket Island. He traveled extensively and was always interested in current events. He had a lifelong fascination for baseball. Only a few days before his death, he predicted the performance of various pitchers and the outcome of the current world series. He was a keen, but an understanding judge of human nature. This, combined with his great sense of responsibility and personal integrity, made John Hughes Dunnington a unique individual, a superb clinician, and a teacher who had great influence on American ophthalmology throughout his career. He represented the ideal of a great physician, and the heritage of ophthalmology is inestimably richer for his devotion. His death is mourned by his colleagues, all of whom were his dear friends.

In 1919 he married Genevieve Richards Parker. Theirs was an enduring and devoted relationship. For over half a century she was his loyal support at all medical meetings and conferences, her gracious presence adding much to the social aspects of those occasions. Our deepest sympathy is extended to her, to their daughter, Jean Cullen, and to their grandson, Louis Coffin.

CHARLES J. CAMPBELL

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on $8^{1/2} \times 11$ -inch bond paper, with $1^{1/2}$ -inch margins on all four sides, and limited in length to two manuscript pages.

Antibiotics in Artificial Tear Solutions: Correction

Editor:

Frederick Theodore recently informed me that reference 1 is incorrect in the article, "The stability of ten antibiotics in artificial tear solutions" (Am. J. Ophthalmol. 82:775, 1976), by Edward Osborn, M.D., Jules L. Baum, M.D., Chaim Ernst, M.D., and Paul Koch, A.B. Dr. Theodore presented this material at the Symposium on Ocular Therapy at the American Academy of Ophthalmology and Otolaryngology in Las Vegas in 1970 and it was subsequently published. The reference should read: Theodore F. W.: Points in the treatment of bacterial corneal ulcers. In Leopold, I. H. (ed.): Symposium on

Ocular Therapy, vol. 5. St. Louis, C. V. Mosby, 1972.

JULES L. BAUM, M.D. Boston, Massachusetts

BOOK REVIEWS

Visual Impairment in Children and Adolescents. By James E. Jan, Roger D. Freeman, and Eileen P. Scott. New York, Grune and Stratton, Inc., 1977. Clothbound, 418 pages, table of contents, author index, subject index, black and white figures, 48 tables. \$29

Few tasks are more difficult than informing parents their child never will have useful vision. Fortunately events rarely make this duty necessary. Since we cannot know when such a patient may appear, we must be prepared for this difficult, but important function. If we fail to dispel all doubts that vision will be possible, the parents will be forced to spend money and energy in the futile search for successful treatment. On the other hand, if the sad conclusion is not transmitted to the parents in a compassionate and appropriate manner, they may react with excessive guilt or even rejection of the child.

Few, if any, sources have been available which adequately give the overall picture of the needs and care of the blind child and his or her family. This void has been filled in an excellent fashion by Jan's book. In spite of heavy involvement with the blind community for several years, I found the book of immense worth and would heartily recommend it as required reading for every ophthalmologist who deals with children. There are a few pearls found in almost every chapter. The modest amount of redundancy from chapter to chapter is not objectionable

since the authors' points made are so important for the proper counseling of the parents.

The authors go to great effort to dispel myths and bias surrounding the blind child. Although a blind child does have a greater than average chance of having other problems, most blind children are capable of developing at a normal pace, if proper attention is devoted to them. A multitude of special problems are present for the parents and teachers of the blind child and we as physicians must be careful not to superimpose further problems. For example, the blind child will often have abnormal electroencephalographic recordings. Care must be taken to not interpret these spikes as epilepsy, for the abnormalities of recordings disappear with age and there is no indication that epilepsy is associated with these findings unless clinical signs of a convulsive disorder are present.

The authors mention certain unexplained associations such as the superior intelligence often found in children with bilateral retinoblastomas.

The book is more than a review of published reports. The authors have made extensive surveys of the Vancouver area to study familial settings of the blind child and compare the attitudes and feelings of parents with control families. The families generally were found to be the same as the control with no increase in divorce or psychological problems. It is noteworthy that the blind child spent much more time in the hospital the first year of life than the normally sighted child.

Reading this book will help most professionals caring for the blind and should especially help the ophthalmologist to advise and counsel the parents of the blind child appropriately.

The authors have written a companion book for parents of the blind child: Can't Your Child See? A Guide for Parents and Professions.

ROBERT D. REINECKE

Eye Surgery: Innovations and Trends, Pitfalls, Complications. Edited By R. M. Fasanella. Springfield, Charles C Thomas, 1977. Clothbound, 325 pages, table of contents, subject index, author index, 25 tables, 83 black and white figures, 25 color figures. \$29.50

The editor and contributing authors address the formidable task of reviewing and analyzing features of many surgical methods, innovations, and instruments recently developed and now widely used. The impeccable credentials of the contributing authors add credibility to the discussion of complex and sometimes controversial methods and procedures. In general, the authors write from broad personal experience, which reflects the optimum capabilities of each new method, although they stress the difficulties and complications that can be encountered. The book is impressively comprehensive although some omissions are obvious-most notably the lack of discussion regarding photocoagulation.

This book reemphasizes the need for continuing education on the part of all ophthalmologists and provides a useful summary of current surgical and medical therapies. Major changes have recently occurred in some aspects of ophthalmic surgery such as widespread use of phacoemulsification, intraocular lens implantation, and pars plana vitrectomy. In other areas such as management of glaucoma and retinal detachment and in oculoplastic surgery, changes have been less dramatic. However, in each area current concepts of pathogenesis and surgical alternatives are summarized in perspective. The varied writing styles of the contributing authors makes for enjoyable reading. However, in some places more careful editing would improve awkard sentence construction and errors of syntax.

Experienced ophthalmic surgeons will find some points of disagreement with the chapter authors and editor. However, the material presented is sound and time will determine the correct point of view. Similarly, methods and innovations described in this book continue to change and already revisions are in order. The next edition most likely will be as valuable as the current one.

RONALD G. MICHELS

Ocular Histoplasmosis. By T. F. Schlaegel, Jr. New York, Grune and Stratton, Inc., 1977. Clothbound, 301 pages, table of contents, index, 64 tables, 136 black and white figures. \$29.50

In his new book, "Ocular Histoplasmosis," T. F. Schlaegel summarizes his own vast experience in this field, combining his observations with the reported data of many other authors. As such, the book represents an important summary of what is currently known or surmised about this baffling malady, aptly referred to as the "presumed ocular histoplasmosis syndrome."

Schlaegel's approach is basically a historical one. After presenting an initial brief chapter on systemic histoplasmosis, the author traces the evolution of various ophthalmologists' findings concerning the ocular syndrome, beginning with Reid's early study in 1942, and progressing to the classic description of the syndrome by Woods and Wahlen in 1959. Ample space is given to the animal experiments, which have supported the hypothesis that Histoplasma capsulatum is the causative organism, but the author is willing to concede that the doubts retained by such workers as George Spaeth are reasonable. Indeed, on pages 36 and 37 he outlines the evidence against H. capsulatum as the etiologic agent. The concept that presumed ocular histoplasmosis syndrome does not represent an inflammation is rejected by Schlaegel who now has histological evidence that inflammatory cells can be found in aggregates or frank nodules at the sites of the fundus lesions. The author is quick to point out, however, that such findings in the routine cases have no connection with the gross inflammatory changes reported by Hoefnagels and Pijpers in a case of *Histoplasma* endophthalmitis. (Chapter 5: Unusual Cases.)

Schlaegel's belief that recurrent inflammation in an old scar incites the breakdown of that scar justifies his advocacy of corticosteroid usage in such cases. Many would disagree with him, relying more heavily on photocoagulation treatment. The rationale for both methods of treatment is discussed; excellent charts, photographs, and diagrams are presented both here and elsewhere in the book.

This can be characterized as a text full of data. Numbers and percentages as well as means and values are constantly quoted. While the author fully realizes the fragility of some of these data (p. 33), such an approach lends confidence to Schlaegel's interpretations and conclusions. The book is a "must" for those who deal with uveitis as a subspecialty. It will be highly useful to many general ophthalmologists as well. The manner of presentation is clear, the bold subtitles help the reader to find the topics of greatest interest; and the reference selection is both appropriate and complete. Because of its usefulness and low price, this book will surely survive for a second edition, an excellent opportunity to correct its many typographical and spelling errors.

G. RICHARD O'CONNOR

SYMPOSIA

Neuro-ophthalmology, vol. 9. Symposium of the University of Miami and the Bascom Palmer Eye Institute. Edited by Joel S. Glaser. Clothbound, 275 pages, table of contents, index, 94 black and white figures, 27 tables. \$37.50

LINDENBERG, R.: How they settled for the calarine cortex

DAVID, N.J.: Amaurosis fugax—and after?

SCHEINBERG, P.: Management of occlusive cerebrovascular disease: a personal approach

DAROFF, R. B.: Evaluation of dizziness and vertigo

TROOST, B. T.: Aneurysms, arteriovenous malformations, and fistulas

RHOTON, A. L., JR., HARRIS, F. S., AND RENN, W. H.: Microsurgical anatomy of the sellar region and cavernous sinus

RHOTON, A. L., JR., AND MANISCALCO, J. E.: Microsurgery of the sellar region

ORR, L. S., SCHATZ, N. J., SAVINO, P. J., AND CORBETT, J. J.: Transsphenodial surgery for large pituitary tumors

ANDERSON, D. R.: Axonal transport in the retina and optic nerve

BIRD, A. C., LEAVER, P. K., GOULD, E., AND MCDONALD, I.: Assessment of intraconal steroids in the treatment of retrobulbar neuritis

PERLMUTTER, J. C., BURDE, R. M., GADO, M., AND ROPER-HALL, G.: Endocrine ophthalmopathy: a disease wearing many masks

SCHMIDT, D.: Saccadic eye movements in myasthenic ocular muscle pareses

Discussions on Glaucoma. Edited by Paul R. Lichter and Douglas R. Anderson. New York, Grune and Stratton, Inc., 1977. Clothbound, 156 pages, table of contents, index, 29 black and white figures, 11 tables. \$16

CHAPTER 1: Diagnostic tests in open-angle glauco-ma

CHAPTER 2: Elevated pressure in the youthful patient

CHAPTER 3: Primary open-angle glaucoma: Decision for therapy

CHAPTER 4: Treatment of primary open-angle glaucoma

CHAPTER 5: Ocular hypertension versus glaucoma

CHAPTER 6: Surgery in primary open-angle glaucoma

CHAPTER 7: Congenital glaucoma

CHAPTER 8: Loss of central vision in glaucoma

CHAPTER 9: Drug side effects

CHAPTER 10: Dealing with a buttonholed flap

CHAPTER 11: Traumatic hyphema and glaucoma

CHAPTER 12: Vitreous loss with filtering surgery

CHAPTER 13: Medical management of acute angle closure

CHAPTER 14: Management after the acute attack

CHAPTER 15: The opposite eye

CHAPTER 16: Differential diagnosis of acute glaucoma

CHAPTER 17: Ciliary block and lens movement glaucoma

CHAPTER 18: Uncontrolled acute angle closure

CHAPTER 19: The asymptomatic narrow angle

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

American Journal of Roentgenology

SCLERAL THICKENING: A COMPUTED TO-MOGRAPHY SIGN OF ORBITAL PSEUDO-TUMOR. Bernardino, M. E., Zimmerman, R. D., Citrin, C. M., and Davis, D. O. (Dept. Radiol., M. D. Anderson Hosp. and Tumor Inst., Houston, Tex.). Am. J. Roentgenol. 129:703, 1977.

Orbital pseudotumor is a benign inflammatory lesion of the orbit. Computed tomography is a new method for evaluation of orbits. Scleral uveal rim thickening with contrast enhancement was found in eight of 15 patients with pseudotumor (53%). Similar rim thickening was seen in one postoperative patient and two cases of recent trauma. Therefore, the sign presumably represents inflammation. It was not seen in 47 cases of thyroid disease and 66 cases of neoplasm. (5 figures, 2 tables, 20 references)—Authors' abstract

ENOPHTHALMOS SIMULATING ORBITAL EMPHYSEMA. Cossrow, J. I., Curtis, J. A., and Edeiken, J. (Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa.). Am. J. Roentgenol. 129: 728, 1977.

Orbital emphysema is a well-documented complication of orbital trauma. Fractures of the ethmoid sinus or maxillary antrum are the most frequent causes, although frontal sinus fractures have occasionally been implicated. A blowout mechanism is usually responsible. We recently encountered a patient in whom severe facial trauma had caused fracture of the nasal bone. Marked enophthalmos produced a radiographic appearance simulating orbital emphysema. This can be distinguished from true orbital air by placing a cotton pledget soaked with water over the globe and repeating the radiograph. If the radiolucent crescent disappears; it is not due to intraorbital air. (1 figure, 4 references)—Authors' abstract

Archives of Neurology

HERPES ZOSTER OPHTHALMICUS WITH CONTRALATERAL HEMIPLEGIA. Pratesi, R., Freemon, F. R., and Lowry, J. L. (Dept. Pediatr., Primeiro Hosp. Distrital de Brasilia, Brazil). Arch. Neurol. 34:640, 1977.

A 48-year-old man developed left hemiparesis nine weeks after herpes zoster skin lesions had appeared over the right forehead. Cerebral angiography showed bilateral changes consistent with cerebral arteritis. The patient's condition worsened after the angiographic procedure. Reports from the literature as well as the present case suggest that arteritis and ischemia best explain contralateral neurological symptoms that occur suddenly following herpes zoster ophthalmicus. (2 figures, 13 references)—Authors' abstract

Archives of Surgery

IPSILATERAL BLINDNESS. A COMPLICA-TION OF CAROTID ENDARTERECTOMY. Treiman, R. L., Bloemendal, L. C., Foran, R. F., Levin, P. M., and Cohen, L. (Cedars-Sinai Med. Ctr., Los Angeles, Calif.). Arch. Surg. 112:928, 1977.

Three patients are described in whom ispilateral blindness developed following carotid endarterectomy. In two cases there appears to have been an occlusion of the central retinal artery and in a third case definite emboli were noted in the retina. All three patients had central scotomas which persisted. The degree of visual loss is not given. The authors note interestingly that two of the three patients had complete occlusion of the internal carotid artery on the ipsilateral side and therefore the embolization must have occurred from the external carotid artery. (4 figures, 19 references)—David Shoch

British Journal of Ophthalmology

CATARACTS AND AVIONIC RADIATIONS. Zaret, M. M., and Snyder, W. Z. (Depts. Ophthalmol. and Physiol., New York Univ. School of Med., New York, N.Y.). Br. J. Ophthalmol. 61:380, 1977.

Avionic radiation refers here to the microwave bands used in radar communication. Bilateral cataracts characterized by selective involvement of the lens capsule were observed in nine patients who had worked for a long time in operational aviation environments containing stray hertzian radiation. Three of the patients were radar technicians serving as in-flight crew on electronic intelligence type aircrafts; five patients were air traffic controllers, and one was an airline pilot. Some of the patients were still able to see 6/6 (20/20) with each eye under the "contrived lighting conditions" of ordinary vision tests, but their performance on the radarscope had become unreliable. In one surgical specimen the inner edge of the lens capsule stood out by its high refractility, and the lens epithelium showed degeneration and vacuolization. Breakdown of lens fibers was most marked in the layers next to the posterior capsule. (3 figures, 16 references)—Peter C. Kronfeld

PRESUMED CHOROIDAL NEVI AND SEN-SORY RETINAL DETACHMENT. Slusher, M. M., and Weaver, R. G. (Bowman-Gray School of Med., Wake Forest Univ., Winston-Salem, N.C.). Br. J. Ophthalmol. 61:414, 1977.

Two white women (in their late 20's) presented with the complaint of blurred central vision and metamorphopsia the cause of which were shallow detachments of the sensory retina over choroidal masses near the fovea, and showing alterations of the retinal pigment_epithelium (including orange pigment) on their inner surfaces. Fluorescein angiography revealed leaks in the area of the detachment. Observation for nine weeks in case 1 and five months in case 2 showed no change in subjective or objective symptoms. The leaks were then treated with low-intensity argon laser photocoagulation. Following the treatment the visual acuity returned promptly to 6/5 (20/15) and the retina reattached over the choroidal masses in both cases. No further change has been noted after four years in case

1 and one and one-half years in case 2. (2 figures, 4 references)—Peter C. Kronfeld

FLUORESCEIN PUPILLARY FLOW IN APHA-KIC EYES WITH AND WITHOUT SPON-TANEOUS OPENINGS IN THE VITREOUS FACE. Zauberman, H., Yassur, H., and Sachs, U. (Eye Dept., Hadassah Univ. Hosp., Jerusalem, Israel). Br. J. Ophthalmol. 61:450, 1977.

The aim of the study was to determine the extent of the adhesions between iris and vitreous face which form during the first four weeks following uneventful intracapsular cataract extractions. The sites of appearance in the anterior chamber of intravenously injected fluorescein were taken to indicate the routes open to and taken by the dye for its passage from the posterior into the anterior chamber. Thirty eyes with peripheral and 30 eyes with sector colobomas were selected for the study. At the time of the test 12 eyes of the former group and 24 eyes of the latter showed openings or rents in the vitreous face (RVF) which were considered to be spontaneous in origin, i.e., not directly related to the surgery. Four variations in the appearance of the dye were observed: (1) only along the pupillary border in eyes with intact vitreous face (32 eyes), (2) only at the pupillary border in the presence of a RVF (10 eyes), (3) only in a RVF (24 eyes), and (4) at the pupillary border and in a RVF (four eyes). Appearance at the pupillary border occurred generally 20 to 30 seconds earlier than in the RVF's; the latter phenomenon was usually associated with visible accumulations of the dye in vitreous pockets. Variations 3 and 4 are interpreted as signs of significant adhesions between iris and vitreous face, possibly the result of forward displacement of the vitreous due to a temporary wound leak for the recognition of which the postoperative observations, apparently, were not close enough. Thus the RVF's may represent new-formed; transvitreal channels for aqueous flow from the posterior into the anterior chamber. By sealing the operative wound more tightly and performing three peripheral iridectomies the authors believe they have significantly reduced the incidence of spontaneous RVF, (4 figures, 2 tables, 9 references)—Peter C. Kronfeld

PSEUDOEXFOLIATIVE DISEASE OF THE LENS: A STUDY IN ELECTRON MICROS-

COPY AND HISTOCHEMISTRY. Dark, A. J., Streeten, B. W., and Cornwall, C. C. (Depts. Ophthalmol. and Pathol., V. A. Hosp., Syracuse, N.Y.). Br. J. Ophthalmol. 61:462, 1977.

Seventeen surgical specimens of cataractous lenses with pseudoexfoliation (PE) were studied to clarify the electron microscopic correlates of the clinical characteristics of PE, i.e., the central disk and the peripheral granular ring on the anterior lens surface. The curledup edges of the two zones prove to be true dehiscences of the superficial capsule for which the term pseudoexfoliation is clearly inappropriate. The central disk is an even layer of fibrils (the characteristic ultrastructural element of PE), intermixed with ovoid bodies which consist of uveal pigment granules, various membrane-bound organelles, and other cell fragments. The peripheral ring is also composed of fibrils and organelles. In the pre-equatorial zone, whole sheets of degenerating iris pigment epithelium, adherent to the lens capsule and the zonules, are a common finding. The fibrils are histochemically similar to but not identical with amyloid. The authors hypothesize that the pre-equatorial epithelial cells of the lens synthesize long-chain, zonule-like proteins which are then converted by lysosomal enzymes, possibly derived from damaged or dying iris pigment epithelium, into small molecules capable of polymerizing within the capsule and in relation to nearby aqueous-bathed structures. Their hypothesis does not preclude the formation of PE fibrils at sites other than the lens capsule. (11 figures, 25 references)-Peter C. Kronfeld

COLCHICINE SUPPRESSION OF CORNEAL HEALING AFTER STRABISMUS SURGERY. Biedner, B. Z., Rothkoff, L., Friedman, L., and Geltman, C. (Dept. Ophthalmol., Soroka Med. Ctr., and Faculty of Health Sciences, Ben Gurion Univ. of the Negev, Beersheva, Israel). Br. J. Ophthalmol. 61:496, 1977.

Two youngsters who had undergone their first extraocular muscle operations by the limbal approach uneventfully, developed after reoperations several years later (again by the limbal approach) corneal dellen which, on conventional therapy, persisted for eight weeks in case 1 and for three weeks in case 2.

When systemic colchicine administration, taken as a prophylactic for familial Mediterranian fever, was discontinued, the dellen healed in two to three days. Since cells with the highest rate of division and metabolism are affected earliest by colchicine, an unfavorable effect of the drug on the healing of the superficial corneal defects is possible. The pronounced swelling of the limbal tissues which often follows the limbal approach after rectus muscle resections, may have acted as an additional disturbing factor. (1 figure, 15 references)—Peter C. Kronfeld

UROKINASE IN THE TREATMENT OF VIT-REOUS HEMORRHAGE. Chapman-Smith, J. S., and Crock, G. W. (Dept. Ophthalmol., Melbourne Univ., and Ophthalmic Research Inst. of Australia, Royal Victoria Hosp., Melbourne, Australia). Br. J. Ophthalmol. 61:500, 1977.

Intravitreal injection of the plasminogenactivator urokinase has been used as the primary treatment for unresolved vitreous hemorrhage and the authors report on the results in 27 patients (34 eyes). The diagnoses were diabetes in 15 patients, systemic hypertension in three, central retinal vein occlusion in two, over-anticoagulation in two, Eales's disease in three and trauma in two patients. Most of the injections were performed under local anesthesia. After release of a small amount of aqueous, 25,000 Plough units of urokinase, freshly dissolved in distilled water, were injected through a pars plana incision under indirect ophthalmoscopic control. More aqueous was released if the eye remained hard. The postoperative course was characterized by transient hypertension and a very striking, but also transient, hypopyon. The injection was repeated once in three patients and twice in one patient. Postoperative observation ranged from 5 weeks to 35 months. The visual results are categorized as follows: (1) improved by more than two Snellen lines (ten eyes), (2) improved by less than two Snellen lines (nine eyes), (3) unchanged (ten eyes), (4) worse (three eyes), and (5) lost to follow-up (two eyes). To the improvement by more than two Snellen lines lens extractions contributed in three eyes and a vitrectomy in one eye. In one of the cases of unchanged vision a vitrectomy with lensectomy also failed to improve vision. Phthisis bulbi ensued after a vitrectomy in one case. Comparing the results of urokinase injections with those of primary vitrectomies for unresolving hemorrhage, the authors conclude that intravitreal urokinase appears to have a higher success rate and a lower complication rate, both in the short and in the long term. Urokinase should be used as "a first line of attack" and vitrectomy be reserved for those patients who fail to improve. (2 figures, 6 tables, 27 references)—Peter C. Kronfeld

Canadian Journal of Ophthalmology

PROGRESSION OF GLAUCOMATOUS FIELD DEFECTS DESPITE SUCCESSFUL FILTRATION. Werner, E. B., and Drance, S. M. (Dept. Ophthalmol., Univ. of British Columbia, Vancouver, British Columbia). Can. J. Ophthalmol. 12:275, 1977.

It is generally assumed that successful filtering surgery, by maintaining intraocular pressure at a low level, will protect a glaucoma patient's remaining visual field. Three patients are presented with chronic open-angle glaucoma and typical visual field changes. In each case, filtering surgery was performed because of progressive loss of visual field and inadequate pressure control. Despite excellent pressure levels after operation, field loss continued in the operated eye. The authors speculate that in the first case, low systemic blood pressure might have contributed to poor perfusion of the optic nerve head, in the second case there may have been vascular changes associated with aging, and in the third patient a combination of aging and low systemic blood pressure may have been responsible for the continuing field loss. In any case it is demonstrated that field loss may progress in glaucoma patients despite good control of pressures following surgery. (9 figures, 3 references)-David Shoch

European Neurology

A CASE OF PROGRESSIVE EXTERNAL OPH-THALMOPLEGIA (KILOH-NEVIN TYPE) WITH ABNORMAL MITOCHONDRIA. Piccolo, G., Cosi, V., Scelsi, R., and Marchetti, C. (Clinica Malattie Nervose e Mentali, Univ. Pavia, Pavia, Italy). Eur. Neurol. 15:325, 1977.

A case of progressive external ophthalmoplegia is described, in which the onset of the illness was at 28 to 30 years, with fatigability and muscular pains in the lower limbs as presenting symptoms. At 36 to 37 years, weakness of the mimic muscles also appeared and fatigability and muscular pains spread to the upper limbs; EMG examinations showed signs of light myopathic involvement of the shoulder-girdle muscles, so that a muscle biopsy was performed (right deltoid). Histoenzymologic studies showed the presence of generally atrophic dark fibers. Ultrastructural study showed bizarrely shaped mitochondria, with dense matrix and circular and confluent cristae, which were found in fibers with plenty of indifferent sarcoplasm and with anomalies in myofibrils. No mitochondrial inclusions were seen. (6 figures, 21 references)-Authors' abstract

Journal of the American Medical Association

HYPERTENSIVE CRISIS WITH BILATERAL BULLOUS RETINAL DETACHMENT. Stropes, L. L., and Luft, F. C. (Dept. Med., Indiana Univ. School of Med., Indianapolis, Ind.). J.A.M.A. 238:1948, 1977.

A 19-year-old man developed blurred vision three weeks prior to admission to the hospital. At the time of admission his vision had diminished to light perception in both eyes. Examination revealed bilateral bullous detachments of the inferior retinas and papilledema. In addition some exudates and small splinter hemorrhages were present. His admission blood pressure was 300/170. With medical control of his hypertension, the vision gradually improved and the retinal detachments subsided. At time of discharge his visual acuity was 6/12 (20/40) in the right eye and 6/8 (20/25) in the left. (4 figures, 5 references)—David Shoch

SICCA SYNDROME IN A PATIENT WITH TOXIC REACTION TO BUSULFAN. Sidi, Y., Douer, D., and Pinkhas, J. (Dept. Med., Bellinson Med. Ctr., Petah-Tikva, Israel). J.A.M.A. 238:1951, 1977.

A 52-year-old housewife had been treated for about ten years with busulfan because of

chronic myeloid leukemia. Some five years after beginning the drug, bilateral posterior subcapsular cataracts were discovered and extracted. Some ten years after starting therapy the patient complained of severe pain in both eyes and dryness of the mouth. Erosions, opacities and engorged blood vessels were seen in the conjunctivas and Schirmer tests confirmed a complete absence of tears. In spite of stopping the drug and the use of artificial tears, the vision was lost in both eyes. (3 references)—David Shoch

Journal of Neurosurgery

ELEPHANTIASIS OF EYELIDS FOLLOWING REPEATED CRANIOTOMY. Amine, A. R., Sugar, O., Patterson, V., and Crouch, E., Jr. (Depts. Neurosurg., Nuclear Med., Ophthalmol., Abraham Lincoln School of Med., Univ. Ill., Chicago, Ill.). J. Neurosurg. 47:293, 1977.

A case is reported in which craniectomy for removal of right frontal meningioma was complicated by porencephaly. A peculiar granulomatous area was found over the brain at reoperation for cranioplasty. Iodine solution was used to paint the reactive material under the scalp flap. Giant swelling of the ipsilateral eyelids gradually developed, ascribed to interference with lymphatic drainage from the eyelids. There was no communication between eyelids and cerebrospinal fluid spaces. The enlarged eyelids were removed by ophthalmic plastic surgery. (3 figures, 7 references)—Authors' abstract

OCULAR BOBBING WITH SUPERIOR CEREBELLAR ARTERY ANEURYSM. Sherman, D. G., and Salmon, J. H. (Divisions of Neurol. and Neurosurg., Southern Illinois Univ. School Med., Springfield, Ill.). J. Neurosurg. 47:596, 1977.

A teen-age girl became comatose after the sudden onset of headache. Initial angiography did not reveal the site of bleeding. The subsequent onset of ocular bobbing directed attention to the region of the pons. Repeated angiography showed an aneurysms of the superior cerebellar artery. At surgery, the fundus of the aneurysm was adherent to the pons and there was a small hematoma within the pons. Ocular bobbing is rare, but is most commonly

seen in association with destructive lesions of the pontine tegmentum, and is a useful localizing sign. (1 figure, 8 references)—Authors' abstract

New England Journal of Medicine

SOCIOECONOMIC FACTORS AFFECTING THE UTILIZATION OF SURGICAL OPERATIONS. Bombardier, C., Fuchs, V. R., Lillard, L. A., and Warner, K. E. (Dept. Health Planning and Admin., School of Public Health, Univ. Michigan, Ann Arbor, Mich.). N. Engl. J. Med. 297: 699, 1977.

Between 1963 and 1970 public programs. were introduced to reduce inequalities in access to medical care. The authors examined differentials in surgical utilization among socioeconomic groups in 1970 as well as changes between 1963 and 1970. Multivariate analysis of National Health Interview Survey data indicated that large increases in surgical utilization occurred among disadvantaged groups: the aged, lower educated and nonwhites in urban areas. Some differential by race and residence remains, but is strongly related to income. Income had a large positive effect on surgical utilization but this effect was less strong in 1970 than in 1963. Among eleven surgical procedures evaluated for complexity, urgency and necessity one was cataract surgery. Physicians generally felt that cataracts did not rate high on the urgency scale but neither did they believe that it is often done unnecessarily. The ranking for cataract surgery in the three categories noted above was third in complexity, 11th in urgency and between second and third in necessity. (4 figures, 3 tables, 15 references)-David Shoch

SULFITE OXIDASE DEFICIENCY. Shih, V. E., Abroms, I. F., Johnson, J. L., Carney, M., Mandell, R., Robb, R. M., Cloherty, J. P., and Rajagopalan, K. V. (Amino Acid Disorder Lab., Massachusetts General Hosp., Boston, Mass.). N. Engl. J. Med. 297:1022, 1977.

At 17 months of age, the child reported here had an episode of vomiting, diarrhea and staggering associated with myoclonic jerks

and ataxic gait. At this time ophthalmic consultation showed only myopia with normal fundi and no lens abnormalities. Ten months later there was an episode of spastic right hemiparesis and weakness of the left side with early spasticity of the left arm. Again, the work-up was negative but no mention is made of the eye examination at this time. At four years of age, ophthalmologic examination revealed subluxation of both lenses, the right being displaced superotemporally and the left lens displaced inferiorly. This caused a reevaluation of his metabolic status and a diagnosis of sulfite oxidase deficiency was then made. This metabolic disorder has been reported only once before and that child too had dislocation of the lenses. It is important to stress that in both the patients there was an absence of homocystinuria. The diagnosis of the first patient reported was made retrospectively after death. The patient here reported is alive and making a good biochemical response to a low sulfur amino acid diet. (4 figures, 2 tables, 28 references)—David Shoch

Ophthalmologica

TUBEROUS SCLEROSIS WITHOUT MENTAL DEFICIENCY OR EPILEPSY. Orzalesi, N., and Grignolo, F. M. (Dept. Ophthalmol., Univ. of Cagliari, Cagliari, Italy). Ophthalmologica 175:241, 1977.

Tuberous sclerosis (Bourneville's disease) is a rare condition characterized by the diagnostic triad of adenoma sebaceum, mental deficiency and epilepsy. The disease is transmitted by a dominant gene with irregular penetrance. Ocular involvement is represented by retinal phakomas which are found in about 50% of the cases. The authors have collected three cases who show typical phakomas of the retina but in none of these was there mental retardation or epilepsy. This is a little unusual. More importantly fluorescein angiography was done on these patients and all showed a lighting up in the early arteriovenous phase which then proceeded very rapidly during the venous phase probably because the extravasation of dye through dilated, abnormally permeable blood vessels. In one case an eye was

enucleated because of pain and the characteristic finding of large numbers of gliocytes was made. There were many dilated blood vessels which would account for the fluorescein picture. (7 figures, 7 references)—David Shoch

SECONDARY GLAUCOMA ACCOMPANIED WITH PRIMARY FAMILIAL AMYLOIDOSIS. Tsukahara, S., and Matsuo, T. (Dept. Ophthalmol., Shinshu Univ. School of Med., Matsumoto, Japan). Ophthalmologica 175:250, 1977.

A group of patients having primary familial amyloidosis was collected in one geographic area in Japan and 15 of these underwent ophthalmologic examination. Secondary glaucoma was found in four of them. In addition to the glaucoma the patients showed pigment deposition in the angle and white flocculate material on the pupillary margin and a flakey substance on the surface of the lens. Antiglaucoma surgery was performed and the specimens showed amyloid fibrils in the iris and trabeculum and it is speculated that these are the cause of the secondary glaucoma. (11 figures, 4 tables, 23 references)—David Shoch

Science

CORNEAL ENDOTHELIUM DAMAGE WITH INTRAOCULAR LENSES: CONTACT ADHESION BETWEEN SURGICAL MATERIALS AND TISSUE. Kaufman, H. E., Katz, J., Valenti, J., Sheets, J. W., and Goldberg, E. P. (Univ. Florida, Gainesville, Fla.). Science 198:525, 1977.

Intraocular lenses destroy corneal endothelial cells by contact adhesion between the acrylic lens and endothelial surfaces during cataract surgery. Glass and rubber surgical glove surfaces produce similar cell damage. This phenomenon may be important in many surgical procedures and appears to be preventable if a hydrophilic polymer interface is interposed between contacting tissue and the surfaces of the materials used. (1 figure, 8 references)—Authors' abstract

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

700 North Michigan Avenue, Chicago, Illinois 60611

For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least three months before the date of occurrence.

GENETIC EYE DISEASES: MEETING

A meeting on "Genetic Disorders Affecting the Eve" will take place at the New Japan Hotel, Tokyo, Japan, May 21, 1978, following the XXIII International Congress of Ophthalmology. Subjects to be discussed include: retinitis pigmentosa, congenital hereditary high blindness, enzyme deficiency and therapy of gyrate atrophy of the choroid, histocompatibility antigens in eye disease, new sphingolipidoses and oligosaccharidoses associated with corneal opacities or retinal disease, eye disorders and chromosomal abnormalities, and inherited macular diseases. Speakers include: Ronald Carr, August Deutman, Edward Cotlier, Eliot Berson, Morton F. Goldberg, and Irene Maumenee. For further information regarding attendance or presentation of free papers, write Ms. Cathy Wingard, Department of Ophthalmology, Yale University School of Medicine, 333 Cedar St., New Haven, CT 065101.

FIRST PHILIPPINE INTERNATIONAL SYMPOSIUM ON CLINICAL OPHTHALMOLOGY

The First Philippine International Symposium on Clinical Ophthalmology, cosponsored by the Far East Graduate Course in Ophthalmology and the Post-Graduate Institute of the New York Eye and Ear Infirmary, will be held in Manila, May 23-26, 1978, following the XXIII International Congress of Ophthalmology in Kyoto, Japan. The honorary chairman of the symposium is Ramon Castroviejo. Byron Smith and Gerald B. Kara are the cochairmen. Dr. Smith will deliver the First Ramon Castroviejo Lecture, "Diagnostic and Therapeutic Trends in Ophthalmic Plastic Surgery." Registration and travel arrangements may be made with Norman Harrison, International Professional Meeting Coordinators, Ltd., 49 West 57th St., New York, NY 10019.

ANGLO-AMERICAN SYMPOSIUM: RESEARCH WITH HUMAN SUBJECTS

The Royal Society of Medicine and the National Institutes of Health, will sponsor a Symposium on Issues in Research with Human Subjects, March 20 and 21, 1978, at the Masur Auditorium, National Institutes of Health, Bethesda, Maryland. For further information, write Mrs. Toby P. Levin, Fogarty International Center, National Institutes of Health, Building 31, Room 2C15, Bethesda, MD 20014; telephone (301) 496-2516.

GERMAN SOCIETY OF OPHTHALMOLOGY: 76TH CONGRESS

The 76th Congress of the German Society of Ophthalmology will be held Sept. 17-20, 1978. The main topics are: ionizing radiation in diagnostics; ionizing radiation in therapy; and radio-isotopies in diagnostics and therapy. Simultaneous translation will be provided in English, French, and German. The deadline for submitting formal applications is March 31, 1978. Preliminary program and application forms are available from the secretariat of Prof. Dr. H. J. Kuchle (Frau Pfeifenberger), Univ.-Augenklinik, Westring 15, D-44, Munster, Germany.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY: NATIONAL SPRING MEETING

The National Spring Meeting sponsored by the West Virginia Academy of Ophthalmology and Otolaryngology will be held April 26-29, 1978, at the Greenbrier Hotel, White Sulphur Springs, West Virginia. Hotel accomodations should be made directly with the Greenbrier Hotel. Check for the registration fee of \$225 should be made payable to West Virginia Academy of Ophthalmology and Otolaryngology, and sent to J. Elliott Blaydes, M.D. Blaydes Clinic, Corner of Frederick and Woodland Avenue, Bluefield, WV 24701.

AMERICAN SOCIETY OF HEAD AND NECK RADIOLOGY, INC.: POSTGRADUATE COURSE •

The American Society of Head and Neck Radiology, Inc., will sponsor a postgraduate course in New York City May 26-28, 1978. Guy D. Potter is pro-

gram chairman. The course will cover all aspects of radiology as applied to head and neck radiology, including routine films, tomography, angiography, ultrasound, and computed tomography. For further information, write Guy D. Potter, M.D., 622 W. 168th St., New York, NY 10032.

OPHTHALMIC SURGERY SEMINAR

A meeting on diagnostic ultrasound for ophthalmology is being co-sponsored by the Vitreo-Retinal Research Foundation, Vitreo-Retinal Service, University of Tennessee, Department of Ophthalmology, Sparta Instrument Corporation, and Xenotec, Ltd. The course director will be Steve Charles. Guest faculty will include Yale Fisher, Jack Kennerdell, Carol Kollarits, James H. Little, F. Hampton Roy, and R. Dudley Stone.

The meeting will be held in Memphis, Tennessee, April 1 and 2, 1978, at the Hilton Inn. Registration fees are: practitioners, \$150; technicians, \$100; and residents, \$50. To make reservations, write: Ophthalmic Surgery Seminars, Ms. Kathleen Noll, 305 Fairfield Ave., Fairfield, NJ 07006; telephone (201) 575-1344.

University of Minnesota; Retinal Disease Course

The Department of Ophthalmology of the University of Minnesota Medical School will present a course entitled "Current Concepts in Retinal Disease" April 17 and 18, 1978, at the IDS Center Conference Theatre, Minneapolis, Minnesota. Guest speakers include: Jerry Shields, Froncie Gutman, Wallack McMeel, Dennis Robertson, and David Elfrig. The course director is Robert C. Ramsay. For further information, write the Office of Continuing Medical Educations, University of Minnesota Medical School, Box 293 Mayo Building, 420 Delaware St., S.E., Minneapolis, MN 55455; telephone (612) 373-8012.

ST. LOUIS MERCY-BAPTIST: EYE SEMINARS

St. Louis Mercy-Baptist will cosponsor its Sixth Eye Seminar, "Highlights of Ophthalmology," with the American Society of Contemporary Ophthalmology, April 2, 1978. For further information, write Eye Seminars, St. John's Mercy Medical Center, 615 S. New Ballas Rd., St. Louis, MO 63141.

HARVARD MEDICAL SCHOOL: NEURO-OPHTHALMOLOGY COURSE

A course in neuro-ophthalmology sponsored by Harvard Medical School will be held April 10-14, 1978, at the Copley Plaza Hotel, Boston, Massachusetts, under the direction of David M. Cogan and Shirley H. Wray. This course is arranged primarily

for ophthalmologists, neurologists, and neurosurgeons. The faculty will include: Daniel Albert, Don Bienfang, Robert Crowell, C. Miller Fisher, Norman Geschwind, John Tittinger, Joel Glaser, Ann Graybiel, David Hubel, Emilio Bizzi, Raymond Kjellberg, Simmons Lessell, W. Ian McDonald, K. Jack Momose, David Newsome, Amy Pruitt, and David Zee. The fee for this course is \$300. For further information and registration, write the Department of Continuing Education, Harvard Medical School, 25 Shattuck St., Boston, MA 02114.

WILLS EYE HOSPITAL AND JEFFERSON MEDICAL COLLEGE: FOURTH ANNUAL CONTACT LENS CONFERENCE

The Fourth Annual Contact Lens conference sponsored by Wills Eye Hospital and Jefferson Medical College will be held June 8-10, 1978, at the Howard Johnson's Regency Hotel in Atlantic City, New Jersey. Registration fee is \$175 for practicing ophthalmologists; \$75 for sponsored technicians; and free for residents who have a letter from their department head. For further information and registration, write Kenneth I. Michaile, M.D., 1930 Chestnut Street, Philadelphia, PA 19103.

ASSOCIATION OF UNIVERSITY PROFESSORS OF OPHTHALMOLOGY, INC.: NEW OFFICERS

The new officers elected to the Association of University Professors of Ophthalmology, Inc., are as follows: Richard O. Schultz, Medical College of Wisconsin, president; David Paton, Baylor Medical College, trustee; George W. Weinstein, University of Texas Medical Center, secretary-treasurer. New members include: Philip D. Aitken, University of Vermont; Budd Appleton, Uniformed Services University of the Health Sciences; David E. Eifrig, University of North Carolina at Chapel Hill; John C. Merritt, Howard University; Manuel N. Miranda, University of Puerto Rico; Joel G. Sacks, University of Cincinnati; Myron Yanoff, University of Pennsylvania; Thom J. Zimmerman, Louisiana State University; John L. Kelter, University of California; Davis and Robert Reidy, University of New Mexico.

The annual meeting for 1978-1979 will be held at Sandpiper Bay, Port St. Lucie, Florida, Jan. 25-27, 1979.

SOCIETY OF AIR FORCE CLINICAL SURGEONS: SEMINAR

The Society of Air Force Clinical Surgeons is sponsoring a surgical postgraduate seminar April 23-26, 1978, at the Biloxi Hilton Hotel, Biloxi, Mississippi. Attendance by civilian physicians is encouraged. For further information, write the pro-

gram chairman, Michael Torma, M.D., HMR Box 1733, USAF Medical Center Keesler, Keesler, AFB, MS 39534.

HARVARD MEDICAL SCHOOL: GLAUCOMA COURSE

Harvard Medical School will sponsor a course in glaucoma April 5-7, 1978, at the Massachusetts Eye and Ear Infirmary under the direction of Paul Chandler, W. Morton Grant, and David K. Dueker. The faculty will include: Daniel Albert, A. Robert Bellows, William Boger, David Campbell, L. Frank Cashwell, Richard Chapman, David Donaldson, David Epstein, Roland Houle, B. Thomas Hutchinson, Thomas Richardson, Richard Simmons, Taylor Smith, David Walton, and Martin Wand. The fee is \$200. For further information and registration, write the Department of Continuing Education, Harvard Medical School, 25 Shattuck St., Boston, MA 02115.

UNIVERSITY OF CALIFORNIA, LOS ANGELES: WASSERMAN PROFESSORSHIP OF OPHTHALMOLOGY

An endowment of one half million dollars from MCA, Inc., Board Chairman and Chief Executive Officer Lew R. Wasserman and his wife, Edie, in honor of Dr. Jules Stein, has established the Wasserman Professorship of Ophthalmology at the University of California, Los Angeles.

ST. MARY'S HOSPITAL, MONTREAL, CANADA: INTRAOCULAR LENS IMPLANT COURSE

An intraocular lens implant course is being offered by St. Mary's Hospital, Montreal, Canada. The two-day intensive course is limited to four or five ophthalmologists and includes direct assisting in the operating room with various intraocular lenses. The course takes place the last Friday and Saturday of each alternative month. For further information, write the Director of Professional Services, St. Mary's Hospital, Lacombe Avenue, Montreal, Canada.

JOINT COMMISSION ON ALLIED HEALTH PERSONNEL IN OPHTHALMOLOGY: NEW OFFICERS

Thomas J. Kirby, Jr., associate professor of ophthalmology at the Mayo Medical School, and chairman of education of the Department of Ophthalmology, was elected president of the Joint Commission on Allied Health Personnel in Ophthalmology during the annual meeting of the American Academy of Ophthalmology. Robert Hugh Monahan is executive vice president; members of the Council are Barnet

Sakler, Peter Y. Evans, Bernard R. Blais, Richard A. Kielar, Budd Appleton, and William E. Scott.

PERSONALS

DOUGLAS R. ANDERSON

Douglas R. Anderson, of the University of Miami School of Medicine, has been named by Research to Prevent Blindness, Inc., as the recipient of its first \$25,000 International RPB-William and Mary Greve Scholars award. In making the award, Dr. Jules Stein, chairman of RPB, cited Dr. Anderson's work in glaucoma pathology as exemplifying "the high standard of vision research which this award has been designed to stimulate and reward."

BERNARD BECKER

Bernard Becker received the Leslie Dana Gold Medal Award from the St. Louis Society for the Blind Nov. 17, 1977. Dr. Becker was cited for his "enormous contribution of time and energy. . . all of which have furthered the ultimate goal of the enhancement of knowledge which will allow the prevention and treatment of blinding diseases."

CLAES H. DOHLMAN

Claes H. Dohlman, professor and chairman of the Department of Ophthalmology at the Harvard Medical School, has been unanimously selected to receive the Distinguished Service Award for excellence in ophthalmology from the American Society of Contemporary Ophthalmology. The Distinguished Service Award, which is the highest honor granted by the American Society of Contemporary Ophthalmology, was presented to Dr. Dohlman by officers of the organization during its 13th Annual Meeting and Scientific Assembly, Jan. 30-Feb. 3, 1978, at the Americana Hotel in Miami Beach, Florida.

HERBERT E. KAUFMAN

Herbert E. Kaufman has been appointed professor of ophthalmology and pharmacology and head of the Louisiana State University Eye Center in New Orleans. Dr. Kaufman assumed this position Jan. 1, 1978.

THEODORE LAWWILL

Theodore Lawwill has been appointed the new editor of Perspectives in Ophthalmology.

Dr. Lawwill, a native of Chattanooga, Termessee, is a graduate of Vanderbilt University in Nashville, Tennessee. After interning at the University of Iowa Hospitals, Iowa City, Iowa, he served his residency at the University of Illinois Research and Education Hospitals and the Department of Ophthalmology, Illinois Eye and Ear Infirmary. Dr. Lawwill is

associate professor of ophthalmology in the Department of Ophthalmology at the University of Louisville School of Medicine.

P. ROBB McDonald

*P. Robb McDonald delivered the 40th Annual deSchweinitiz Lecture on Nov. 17, 1977, at the College of Physicians and Surgeons in Philadelphia. His speech was entitled, "Evolution of cataract surgery since the deSchweinitz era."

HOWARD SCHATZ

Howard Schatz of San Francisco gave the first

James E. McDonald Lecture at Loyola University Stritch School of Medicine Nov. 23, 1977. Dr. Schatz's lecture was entitled, "Hamartomas and Hamartias of the Fundus."

HAROLD G. SCHEIE

Harold G. Scheie, founding director of the Scheie Eye Institute at the University of Pennsylvania, has been unanimously selected to receive the International Glaucoma Congress Medal of Achievement in recognition of his outstanding contributions to glaucoma research, diagnosis, and therapy. The Medal of Achievement is the highest honor granted by the International Glaucoma Congress.

VISUAL FIELD CHARTS

Central and peripheral field charts are available without charge to authors who require them to illustrate their papers. Write to Editorial Correspondent, American Journal of Ophthalmology, 233 East Ontario Street, Suite 1401, Chicago, Illinois 60611.

INSTRUCTIONS TO AUTHORS

For the preparation of manuscripts for American Journal of Ophthalmology

THE AMERICAN JOURNAL OF OPH-THALMOLOGY publishes original and timely contributions dealing with clinical and basic ophthalmology. Each article submitted is evaluated by two or more referees who recommend that the paper be (1) accepted unchanged, (2) returned for revision and subsequent editorial consideration, or (3) rejected. Acceptance is conditioned by such factors as the originality, significance, and soundness of the contribution; the suitability of the subject matter for subscribers of THE JOURNAL; and, the care with which the manuscript has been prepared.

Papers are accepted on the condition that they have not been published or accepted for publication in any other journal, whether printed in English or any other language. On occasion, a paper read before a society and published in the society's transactions will be considered if the society publication does not reach as wide an audience as the contribution merits. When submitting such a paper,

the author must indicate the time and place of the meeting and the name of the society publication. It is not possible to coordinate the date of publication of such papers in THE JOURNAL with that in the society publication.

Authors will be advised promptly of receipt of their papers. Thereafter they will be advised within 30 days of acceptance, rejection, or need for revision. Manuscripts that require extensive editorial correction or retyping will be returned for that purpose.

MECHANICAL PREPARATION OF MANUSCRIPT The manuscript should be prepared in the style used by THE JOURNAL. A heavy grade of white-bond paper measuring 8½ by 11 inches should be used. Margins should be at least ½ inches on all sides. Paragraphs should be indented at least one-half inch. Two copies must be submitted; carbon copies and machine-duplicate copies are acceptable only as second copies.

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The entire manuscript should be double- or triple-spaced. Single-spacing should not be used anywhere in the manuscript. The entire manuscript—including title page and footnotes, text, acknowledgments, references, tables, and legends—should be double-spaced. Rules and underlining should not be used anywhere in the manuscript.

The manuscript should be arranged in the following order:

- 1. Title page (with footnotes)
- 2. Text and summary
- 3. Acknowledgments
- 4. References
- 5. Tables
- 6. Legends for figures

Each major section should begin on a separate sheet.

TITLE PAGE The title page should be numbered page 1 and contain the running (abbreviated) heading, the title, each author's name and highest degree, and the city and state where the work was carried out. The institution and the organization(s) sponsoring the study should be credited in a footnote. A second footnote should give the name and mailing address of the author to whom correspondence should be directed. Each page after the title page should show the page number, senior author's name, and running title in the upper righthand corner.

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Following a description of material and methods, the results of the study should be given. A section devoted to discussion should follow. The discussion should relate directly to the topic of the paper.

Summary—Each paper must have a summary that describes the content of the paper in no more than 150 words. The author should state precisely what was accomplished, and avoid generalities.

Acknowledgments—Sponsoring organizations and grants should be acknowledged in the first footnote on the title page. Other acknowledgments follow the summary.

REFERENCES The author is responsible for complete and accurate references, including the proper capitalization and accent marks used in foreign-language publications. References must be numbered consecutively, according to their appearance in the text. Ex-tensive reviews of the literature are not desirable. Personal communications should be kept to a minimum. Reference to studies that have been accepted for publication, but not yet published, should indicate where they will be published. Reference to studies still in progress should be described as such in the text without a reference number. Primary, not secondary, sources should be cited; references derived from encyclopedic reviews or textbooks are seldom acceptable. References should be cited in the text as follows: Allen and Smith1 and Jones2 described . . .

The names of all authors should be cited in the reference list. THE JOURNAL does not use the term et al. The following

Instructions to Authors—CONT.

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1. Terry, T. L.: Extreme prematurity and fibroblastic overgrowth of persistent vascular sheath behind each crystalline lens. Am. J. Ophthalmol. 25:203, 1942.

2. Reese, A. B.: Tumors of the Eye, 2nd ed. New York, Hoeber, 1964, p. 91.

Abbreviations of periodicals are listed in *Index Medicus*. If there is any question, the name of the publication should be written out in its entirety.

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Fig. 1 (Jones, Smith, and Brown). Histologic section of the eye (hematoxylin and eosin, ×70).

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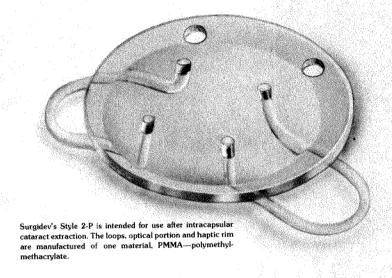
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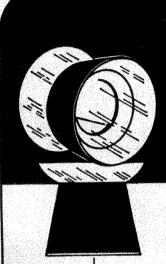
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British Journal of Ophthalmology

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Editorial: Ocular hypertension

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Robert David, Desiree G. Livingston and Maurice H. Luntz

Diathermo-trabeculotomy ab externo: indications and long-term results. E. Maselli, G. Galantino, F. Pruneri and M. Sirellini.

External ocular motor palsies in ophthalmic zoster: a review. R.J. Marsh, B. Dulley and V. Kelly

Structural alterations of extraocular muscle associated with Apert's syndrome. Sheila Margolis, Bruce R. Pachter and Goodwin M. Breinin.

Diagnosis and measurement of cyclodeviation. D.K. Sen, B. Singh and N.M. Shroff.

Unusual ocular presentation of acute toxoplasmosis. Darrell Willerson, Jr., Thomas M. Aaberg, Frederick Reeser and Travis A. Meredith.

HLA-B27 frequency in Greek patients with acute anterior uveitis. J. Zervas, G. Tsokos, G. Papadakis, E. Kabouklis and D. Papadopoulos.

Bull's eye maculopathy with early cone degeneration. R.H.B. Grey, R.K. Blach and W.M. Barnard.

An unusual presentation of Best's disease. Robert C. Fletcher, Lee M. Jampol and William Rimm.

An improved objective slit-lamp fluorophotometer using tungsten-halogen lamp excitation and synchronous detection. A. Trevor-Smith, D.P. Jones, G.D. Sturrock and Peter Wright.

A cannulated probe for torn inferior canaliculus repair. Peter T.C. Docherty.

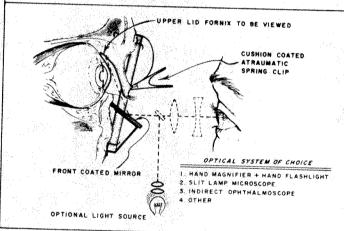
Rapid deterioration of visual fields during bromocriptine-induced pregnancy in a patient with a pituitary adenoma. J.T.W. Van Dalen and E.L. Greve.

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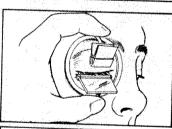


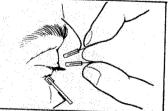
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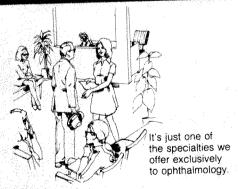
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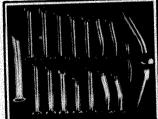


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Dates:

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Dates:

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WHEN THE DOCTOR DIES

You will probably need to know these items:

- 1. Social Security Number or Taxpayer's Identification Number
- Accountant or Estate Planner: Name, address, phone number
- Attorney: Name, address, phone number
- Employee(s): Name(s), address(es), phone number(s), details of termination of services

 5. Insurance Agent (and/or Estate Planning Man-
- ager): Name, address, phone number 6. Stockbroker: Name, address, phone number 7. Trust (if established): Name of Trustee, address.
- phone number, name of bank's representative, phone, extension if the trustee is a bank Will: Location, location of any codicils
- 9. Home: Location of deed, mortgage papers, etc.
- Office: Location of deed or lease, mortgage papers, duration of lease or mortgage, provision for settling in event of death
- 11. Other real estate: Location(s) of deed(s) or mortgage paper(s)
- 12. Life Insurance: Location of policies
- Other Insurance: Location of policies
- Temporary coverage of the Practice: Name, address and phore number of an associate
- 15. Securities: Location of policies 16. Current unpaid bills: Location
- Outstanding obligations: Notes, mortgages, pledges, charge accounts, credit checking acmortgages, counts, etc. Details
- 18. Safety Deposit Box(es): Location(s), number(s), location of keys
- Savings Account(s): Location(s) of passbook(s)
- 20. Checking Account(s): Location(s), location(s) of checkbook(s)
- Financial Records: Location
- Cash: Location of receipts, unbanked cash, source of cash for immediate needs
- 23. Tax Information: Location
- 24. Accounts Receivable: Location, the Doctor's views on collecting current and delinquent bills
- 25. Notes, loans, mortgages, etc., due from others: ocation of items
- 26. Notifying patients: Method preferred by the doctor, location of appointment book
- 27. Notification: Hospital(s)—(Administrator), phone numbers: colleagues, phone numbers, Societies, etc., phone numbers
- 28. Veterans Administration: Branch of Service, date of discharge, service number, VA number(s), location of discharge papers
- 29. Blue Cross, etc.: Policy numbers of Blue Cross, Blue Shield and other Health Insurance (see later discussion)
- 30. Narcotics: Location of drugs, tax stamp, narcotics ledger, narcotics order book
- Equipment (major): Purchase price, sales outlet
- The Doctor's views on disposition of property, securities, anticipated income, etc.
- 33. Birth and Marriage Records: Location
- 34. Citizenship Papers (if applicable): Location

In addition to the foregoing but subject to the rights and duties of the Executor or Administrator, you may become involved with the following:

- I. Probate of the Will: Your attorney will do this.
- II. Disposition of narcotics, drug samples and medications. You should contact your Regional Office of the Bureau of Narcotics and Dangerous Drugs, U.S. Department of Justice, requesting disposition forms which must be completed and returned.

III. Patient Records:

- A. These are important and confidential records which should be carefully preserved. Apparently there is no set limit of time specified by
- B. The contents of such records should be disclosed to another physician only if the patient so requests. Such a request should be in writing and should be retained with the records. Normally, the records are confidential and diagnostic information in them should not be disclosed directly to the patient because of the risk that he might misinterpret them.
- C. All patients' medical records should be kept for a minimum of ten years. Records of patients who were minors when treated should be preserved until two years after the patient reaches twenty one. When possible, records should be kept for twenty-five years. It should be kept in mind that requirements for the retention of medical records may vary from state to state. Accordingly, before any medical records are destroyed or disposed of, local requirements should be verified with either the attorney for the estate or the local medical society. Financial records should be kept for five years, which is the statute of limitations on such information.
- IV. Telephone: If there is an answering service, this can be a very helpful way of notifying patients of the Doctor's death with possible suggestions regarding their continued care. It is suggested that this service be retained for at least a month.
- V. Notifying patients: Perhaps a simple sign on the office door "Call (office phone number)" will relieve you of the burden of contact with most of the patients and the answering service can take care of the matter.

If the physician's secretary is retained temporarily she might perform this service by being present during usual office hours. She also could notify patients with future appointments during this time.

VI. Taxes: The accountant and/or attorney should take care of all of this in connection with the financial records.

VII. Stopping advertising mail: It may take a year for knowledge of the Doctor's death to reach all the mailing lists; there is no central clearing office. You may perhaps choose to let it run out in this fashion, or you may write "Deceased return to sender" on each piece and mail it back. While the latter course might entail some effort initially, it will rapidly and effectively cut off the flow.

VIII. Insurance Agent and/or Trustee: A preliminary brief conference might be in order to settle the question of immediate funds, documents to be located, and arrangements for a later, detailed conference. Prompt notification of such people is important.

IX. Consideration of Sale of Practice: If the practice is to be sold, with or without real estate, relatively rapid action is advised, for it will quickly lose value. It can be advertised in the State Medical "Journal" and the "Journal" of the American Medical Association.

X. Real Estate: Except for practice-connected real estate, a hasty decision on sale or retention is not advised. It is hoped that the doctor has made known his views on this subject.

XI. Temporary coverage of patients:

- A. Some arrangement with a colleague should be made immediately as regards patients in the hospital.
- B. While not required, it would be a courtesy to be able to suggest a colleague to other patients.

XII. Insurance:

- A. Malpractice Insurance: A refund on unused premiums may be possible. Find the policy and consult the local agent.
- B. Liability and other Insurances: Certain policies issued with reference to the practice might have provisions for refund of unused premiums. Consult the issuing company or your insurance agent.
- C. Health and Hospital Insurance: The survivor of a physician who had hospital or other health insurance under a group or family plan may keep such insurance, but it should be transferred to individual instead of family policies, unless you have dependent children.
- XIII. Veterans Administration: If the Doctor was a Veteran, notify the Veterans Administration with the indicated information and apply for deserved benefits. Help with this can be had through the Veteran's Service Officer of the Doctor's county or district, or the Veterans Administration Regional Office.

In this connection, save the Doctor's discharge papers permanently. It is possible that Pensions or other forms of remuneration may be provided in the future.

XIV. Social Security: Nearly all physicians are now covered by the Social Security Act. Visit the Social Security Office serving your area to find out what benefits are available to you. Be certain to have the Doctor's Social Security or Taxpayer Identification Number.

XV. Automobile(s): Title, if in Doctor's name, can be transferred with assistance of an automobile club, if a member, or through the Department of Motor Vehicles. Be sure insurance coverage is re-

XVI. Equipment: It is best disposed of as part of the practice, if possible. Otherwise, it might be possible to obtain an estimate of value from one of the reputable equipment vendors and then sold either privately, through an advertisement in the State Medical "Journal" or to a dealer of used equipment. One of the Doctor's colleagues can give you the names of the major supply dealers serving your area.

XVII. Current bills: These must be paid, but not until the court has approved the Executor or appointed the Administrator of the Doctor's estate.

XVIII. Magazine subscriptions: Those purchased for office use might be checked for possible stoppage and subscription refund if you do not wish to continue them.

XIX. Dues: There is ordinarily no refund on Medical Society dues unless hardship exists.

XX. Securities: It is worth noting that Securities can be used as collateral for loans to meet financial needs. This may be preferable to selling the securi-

The American Society of Internal Medicine has learned with regret of the Doctor's death. We wish to extend our condolences. We realize that you will have many tasks in connection with settlement of the estate. To assist you the Society has prepared this pamphlet.

It should be pointed out that many of the items appearing herein are matters which, technically, will be the professional responsibility of the Executor (if the Doctor died with a Will) or the Administrator (if the Doctor died without a Will). However, the information contained herein may prove to be helpful not only to the person having this responsibility but also to the Doctor's survivor. We recommend that the survivor discuss the contents of this pamphlet with the Executor or Administrator of the Doctor's estate as soon as possible.

REPRINTED WITH PERMISSION FROM THE AMERICAN SOCIETY OF INTERNAL MEDICINE.

THIS BROCHURE WAS PREPARED BY THE NORTH CAROLINA SOCIETY OF INTERNAL

MEDICINE AND THE CLEVELAND (OHIO) ACADEMY OF MEDICINE.

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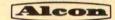
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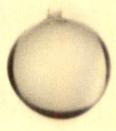
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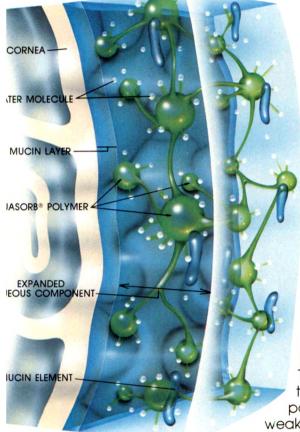
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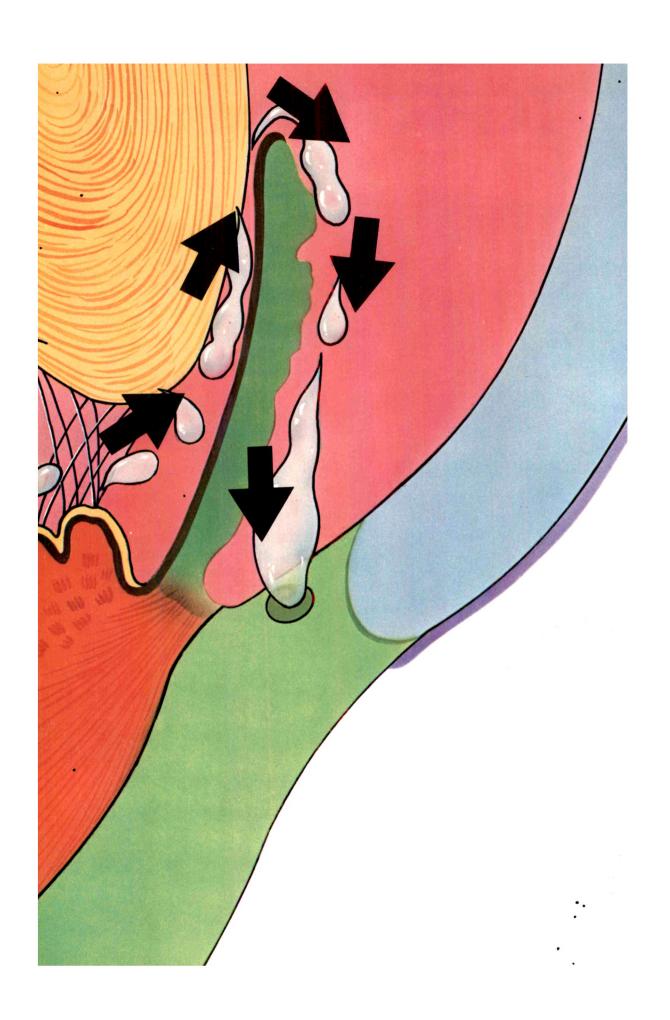
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AMERICAN JOURNAL OF OPHTHALMOLOGY

SERIES 3

VOLUME 85

NUMBER 6

IUNE 1978

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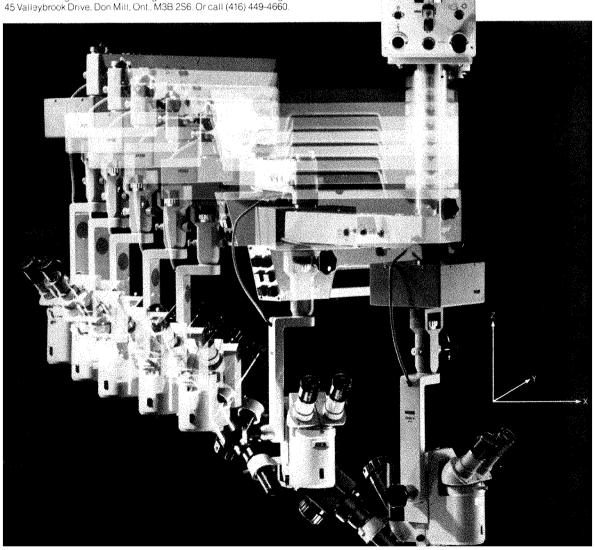
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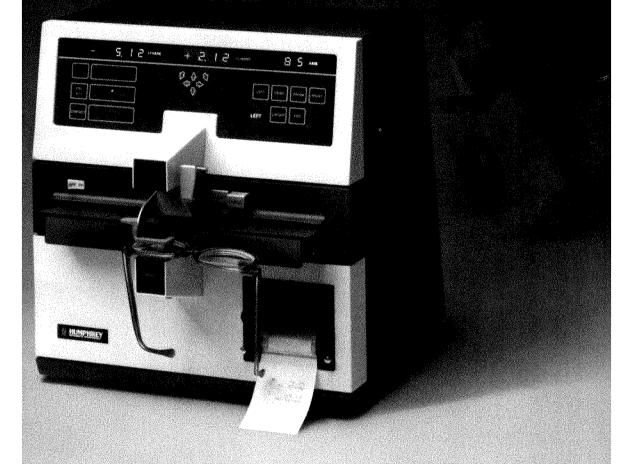
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Now you can spend your valuable time on more important things. You can increase your practice income potential, make your practice more cost-efficient, increase your patient flow. Today, thousands of your colleagues are enjoying less complicated practices by building on the AOSOFT lens, the uncomplicated soft contact lens. Shouldn't you make it the primary lens in your practice, too?

1 Data on file. American Optical Corp., Soft Contact Lens Div. Southbridge, MA 01550





See next page for prescribing information.

AOSOFT® (tetrafilcon A) Hydrophilic Contact Lenses DESCRIPTION

DESCRIPTION

AGSOFT* (letrafilicon A) Hydrophilic Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sciera. The lens material, tetrafilicon A, is a hydrophilic random terpolymer of 2-hydroxyethyl methacrylate, N-vinyl-2-pyrolidone and methylmethacrylate. The polymer is a three-dimensional network of terpolymer chains joined by divinyl-benzene cross-links. It consists of 57.5% tetraficion A and 42.5% water by weight when fully hydrated in 0.9% sodium chloride solution buffered with moschalars and filhasic sodium phosphates. monohasic and dibasic sodium phosphates.

ACTIONS

ACTIONS
In its hydrated state, the AOSOFT* Hydrophilic Contact Lens is soft and pliable. When dry, the lens becomes hard and brittle. These states are completely reversible and a lens which has been permitted to dry out will recover all of its hydrated properties when placed in normal saline for a period of two hours. When placed on the human cornea, the hydrated lens acts as a or two mous. When placed on the numan comea, the hydrated lens acts as a refracting medium to compensate spherical ametropias. The material has a refractive index of 1.43 and the lens has a visible light transmittance greater than 97%.

INDICATIONS

AOSOFT* Hydrophilic Contact Lenses are indicated for the correction of vision in persons with non-diseased eyes who have spherical ametropias, corneal astigmatism of 2.50 diopters or less and/or refractive astigmatism of 2.00 diopters or less.

CONTRAINDICATIONS

AOSOFT⁸ Hydrophilic Contact Lenses are contraindicated in the presence of any of the following conditions: (1) Acute and subacute inflammation of the anterior segment of the eye. (2) Any eye disease which affects the cornea and conjunctiva. (3) Insufficiency of lacrimal secretion. (4) Corneal hypoesthesia. (5) Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses

WARNINGS

WARNINGS
Medications and Eye Drops: AOSOFT* Lenses must be stored in an appropriate solution when off the eyes, the type of solution being dependent on the system used for disinfection. When the lenses are disinfected with the thermal disinfection system, they may be stored only in LENSRINS* or BOIL in SOAK* Solution (sterile, buffered preserved saline solution). When lenses are disinfected with the chemical disinfection system, they may be stored only in FLEXSOL* Solution (sterile, isotonic buffered solution containing Adsorbobase — a water soluble polymeric vehicle— and preserved with themerosal 0.001%, chlorhexidine 0.005%, and edetate disodium 0.1%).

Soluble polyments vehicle—and preserved with finnersolar doorns, clinichrekaws 0.005% and edetate disodium 0.1%). No ophthalmic solutions or medications, including hard contact lens selution, can be used by AOSOFT* Lens wearers prior to or while the lenses are inplace on the eyes. Also, no solutions, including hard contact lens solutions, other than LENSRINS* or BOIL n SOAK* Solution or FLEXSOL* Solution may be used on AOSOFT* Lenses when they are off the eyes. Since liquids and vapors may be absorbed by AOSOFT* Lenses, they should not be piaced in the mouth for wetting, nor wiped with a cloth or tissue. Abrasions and Infections: It a lefts becomes less comfortable than it was when first piaced on the wearer's cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body If any eye abrasion, ulceration, irritation or infection is present, or any abnormal eye condition is observed concurrently with lens wear. The lens should be removed immediately and a physician consulted. Wearing Restrictions: AOSOFT* Hydrophilic Contact Lenses should not be worn while swimming, sleeping, or in the presence of irritating tumes or vapors. Visual Blurring: If visual blurring occurs, the lens must be removed until the condition subsides.

condition subsides.

Lens Sanitation: Patients must adhere to the recommended daily sanitary care procedures for AOSOFT* Hydrophilic Contact Lenses Failure to follow this procedure may result in the development of serious ocular infections.

PRECAUTIONS

Storage: AOSOFT* Lenses may be stored only in the appropriate storage solution. LENSRINS* or BOIL n SOAK* Solution or FLEXSOL* Solution depending on disinfecting methods used. If left exposed to air, the lenses will dehydrate, become brittle and break readily if a lens dehydrates, it should be soaked in either LENSRINS*, BOIL n SOAK* or FLEXSOL* Solution until it returns to a soft, supple

Cleaning and Disinfecting: AOSOFT* Lenses must be both cleaned and

Cleaning and Disinfecting: AOSOFT* Lenses must be both cleaned and disinfected daily Separate procedures and products are needed to clean and to disinfect two methods of disinfection, thermal or chemical, have been shewn to be equally effective. The choice of disinfection system should be made in consultation with your eye care practitioner.

Cleaning: Daily cleaning is necessary to remove mucus and other deposits which may have accumulated on the lens surface. Each time the lenses are removed from the weare's eyes, both surfaces of the lenses must be cleaned using several drops of PREFLEX* Cleaning Solution (sterile solution of a nonionic cleaner preserved with thimerosal 0.002% and debtare disodium 0.1%). Lenses must be cleaned before they are disinfected, as deposits on the lenses tend to harden and become more difficult to remove after the lenses are disinfected.

Disinfecting: AOSOFT* Lenses may be disinfected with either a thermal or chemical regimen. One method or the other must be selected, but not both THE USER MUST NOT ALTERNATE BETWEEN METHODS.

Thermal Disinfection Method: AOSOFT* Lenses may be effectively disinfected after cleaning with PREFLEX* Cleaning Solution with use of the AOTHERM* Heating Unit For Lens Disinfection and LENSRINS* or BOIL in SOAK* Solution Fresh LENSRINS* or BOIL in SOAK* Solution pust prior to disinfecting the lenses. prior to disinfecting the lenses.

Chemical Disinfection: Disinfection with PREFLEX® Cleaning Solution, FLEXSOL® Solution and NORMOL® Rinsing Solution (sterile isotonic saline solution preserved with thimerosal 0.001% and chlorhexidine 0.005%) has also been shown to be an effective disinfection system for daily care of AOSOFT

been shown to be an effective distinlection system to duity care or ACSOFT*
Lenses. AOSOFT* Lenses must be cleaned and finsed daily (or after wearing) with
PREFLEX* Cleaning Solution and NORMOL* Rinsing Solution. The AOSOFT*
Lens Storage Containers must be emptiled and refilled with fresh FLEXSOL*
Solution each time the lens is stored. Fresh FLEXSOL* Solution must be used daily for storage and disinfection.
WARNING. DO NOT MIX OR ALTERNATE THE DISINFECTION AND STORAGE
SYSTEMS FLEXSOL* SOLUTION SHOULD NOT BE USED WITH HEAT.

Hygiene: Before handling the lenses, hands must be washer, rinsed thoroughly and dried with a lint-free towel. Cosmetics, lotions, soaps, oil and hand creams must not come in contact with the lenses since eye irritation may result, if hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Thurner Spray has section.

Fluorescein: Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with LENSRINS* or BOIL in SOAK* and wait at least one hour before. replacing the lenses. Too early replacement may allow the lenses to absorb residual fluorescen.

ADVERSE REACTIONS

Serious corneal damage may result from wearing lenses which may have soaked in hard contact lens solutions. Eye irritation may occur within a sh time after putting on a hypotonic lens. Removal of the lens will relieve the irritation

Irritation. Very rarely a lens may adhere to an eye as a result of a patient sleeping with the lens on, or as a result of wearing a hypotonic ens. If a lens adheres for any reason, the patient may be instructed to apply a lew drops of LENSRINS or BOIL in SOAK* Solution (if using a thermal disinfection regimen) or ADAPETTES* Cleaning and Rewetting Solution (sterile, isotonic, buffered solution containing Adsorbobase and preserved with thimerosal 0.002% and edetate disordium 0.05%) (if using a chemical disinfection regimen), and wait until the lens moves freely before removing it.

Clinical studies indicate that corneal edema as manifested by symptoms with a sembours or abore around their own and approprint flores.

Clinical studies indicate that corneal edema as manifested by symptoms such as rainbows or halos around light or visual burring may occur if lenses are worn continuously for too long a time. Removal of the lenses and a rest period of at least one hour generally relieve these symptoms. If symptoms do not subside promptly, professional consultation should be obtained. Excessive tearing, unusual eye secretions and photophobia are not normal; if these symptoms occur, the patient should be examined to determine their cause.

POSAGE AND ADMINISTRATION
Fitting: Conventional methods of fitting contact lenses do not apply to
AOSOFT* (tetrafiicon A) Hydrophilic Contact Lenses: For a detailed description of
the fitting technique, refer to the Fitting Guide for AOSOFT* Hydrophilic Contact
Lenses, copies of which are available from: American Optical Corp., Soft Contact
Lens Div., Southbridge, MA 01550.
Wearing Schedule: There may be a tendency for the patient to overwear the
innerse institute Therefore, the importance of adhering to the Editowico instituction.

lenses initially. Therefore, the importance of adhering to the following initial daily wearing schedule should be stressed to the patient.

Day	Wear Time (hours)	Rest Period (hours)	Wear Time (hours)
1	4	2	4
2	4	2	4
3	5	2	5
4	6	2	5
5	7	2	5
6	7	1	6
7	8	1	7
8	8	1	8
9	9	1	8
10-14	10	1	balance of
15	ail waking hours		waking hours*

*lenses should never be worn 24 hours a day

Lens Care and Handling: Care must be taken on the initial visit to assure that the patient is supplied with an AOSOFT* Wearer's Kit and fully understands all care and handling instructions for the lenses. As with any contact lens, regular recall visits are necessary to assure patient health and compliance with

How Supplied: Each lens is supplied sterile in a glass vial containing 0.9% sodium chloride solution buffered with monobasic and dibasic sodium phosphates. The glass vial is marked with the Vault Number, Dioptric Power, and Lot Number.

The AOSOFT* Wearer's Kit is required for lens cleaning, disinfection and storing.

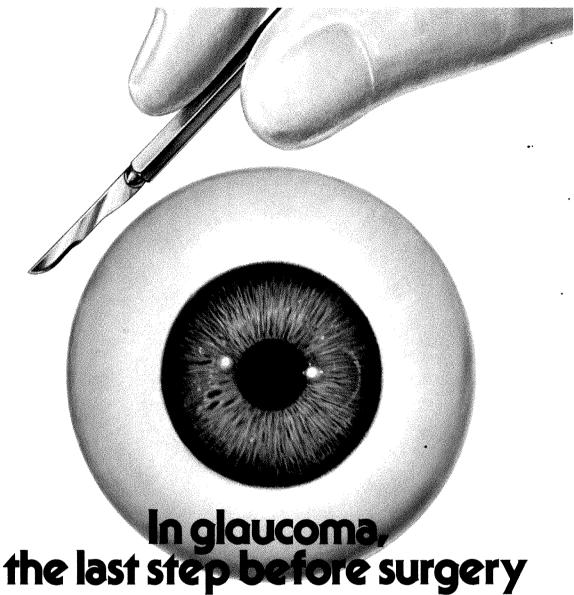
The AOSOFT* Wearer's Kit is required for lens cleaning, disinfection and storing of the lenses. The Kit may consist of either of the following:

Thermal Disinfection Regimen: AOSOFT* Wearer's Kit, AOTHERM * Heating Unit For Lens Disinfection, LENSRINS* Solution, PREFLEX* Cleaning Solution, AOSOFT* Wearer's "How To" Book, AOSOFT* Lens Storage Container:

Chemical Disinfection Regimen: AOSOFT* Wearer's Kit, AOSOFT* Lens Storage Container, PREFLEX* Cleaning Solution, NORMOL* Rinsing Solution, FLEXSOL* Storage and Disinfection Solution, AOSOFT* Wearer's "How To" Book, PREFLEX, NORMOL, FLEXSOL, ADAPETTES, and BCIL in SOAK are registered trademarks of Button, Parsons & Company Inc. marks of Burton, Parsons & Company, Inc

CAUTION: Federal law prohibits dispensing without prescription





When chronic glaucoma becomes refractory to acetazolamide and miotics, consider NEPTAZANE before resorting to surgery. A significant number of patients no longer responding to acetazolamide have been controlled by methazolamide! There are differences, too, when it comes to side effects, since many patients intolerant to acetazolamide may be maintained on methazolamide. So whether the problem is inadequate I.O.P. control or drug intolerance, NEPTAZANE is worth a trial before surgery.

Before prescribing, please consult complete, product information, a summary of which follows:

Indications: For adjunctive treatment of chronic simple (open angle) glaucoma, secondary glaucoma, and preoperatively in acute angle clasure glaucoma where delay of surgery is desired in order to lower intraocular pressure.

Contraindications: Severe or absolute glau-Controldications: Severe or absolute glau-coma and chronic noncongestive angla-closure glaucoma. Of doubtful use in glau-coma due to severe peripheral anterior synechiae or hemorrhagiic glaucoma Adrenocorricol. hepanic or renol insufficiency: electrolyte imbalance state. e.g., hyper-chloremic acidosis: sodium and potassium depoletion strats. dealetion states

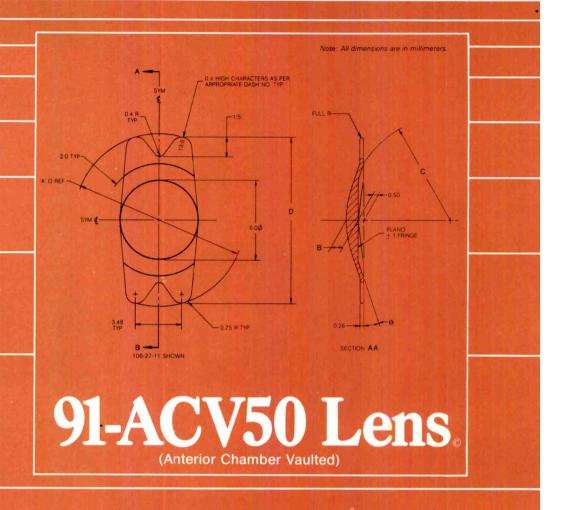
Warnings: Although teratogenic effects demonstrated in rats at high doses have not been evidenced in humans. Methazolamide should not be used in women of childbearing potential or in pregnancy, especially in the first trimester, unless the expected benefits outweigh potential adverse effects.

Precautions: Use with caution in patients with cirrhosis or hepatic insufficiency to forestall hepatic coma: those on steroid therapy: those with pulmonary abstruction or emphysema to avoid acidosis. Electrolyte balance should be maintained. Although not reported thus far with this drug, reactions common to sulfon-amide derivatives, such as fever leukopenia hemolytic anemia, bone morrow depression or renal calculi, may occur

Adverse Reactions (relatively mild and disappear on withdrawal or dosage adjustment): anorexia, nausea, vomiting malaise, fatigue or drowsiness, headache vertigo, mental confusion, depression, paresthesias. Urinary citrare excretion and uric acid output is decreased during use of this drug, but unnary calculi have not been reported







We know that in every surgical situation the minimization of risk to the patient is paramount in importance to the physician.

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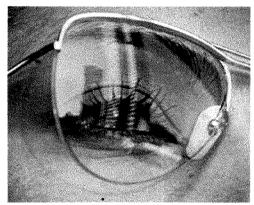


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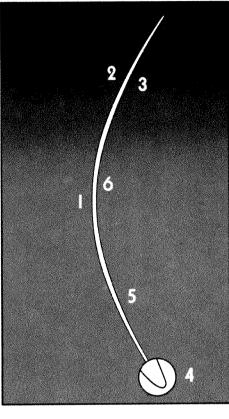
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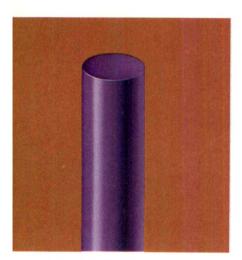
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ETHICON the closing word



COMPLETE PRODUCT INFORMATION

VICRYL* (Polyglactin 910) Synthetic Absorbable Suture

DESCRIPTION VICRYL (polyglactin 910) synthetic absorbable suture is prepared from a copolymer of glycolide and lactide. These substances are derived respectively from glycolic and lactic acids. The empirical formula of the consultance if CHO in CHO Inc. polymer is $(C_1H_1O_2)m(C_1H_1O_2)n$.

VICRYL sutures are sterile, inert, nonantigenic, nonpyrogenic, and elicit only a mild tissue reaction during absorption. The braided and monofilament sutures are colored violet to enhance visibility in tissue. The braided suture is also available under the colored violet to enhance visibility in tissue. able undyed (natural).

ACTIONS Two important characteristics describe the *in vivo* behavior of absorbable sutures: first, tensile strength retention, and second, the absorption rate (loss of mass). Subcutaneous tissue implantation studies of VICRYL suture in rats show at two weeks post-implantation approximately 55% of its original tensile strength remains, while at three weeks approximately 20% of its original strength is retained. Intramuscular implantation studies in rats show that the

absorption of ViCRYL suture is minimal until about the 40th post-implantation day. Absorption is essentially complete between the 60th and 90th days. INDICATIONS VICRYL synthetic absorbable suture is intended for use as an absorbable suture or ligature.

CONTRAINDICATIONS This suture, being absorbable. should not be used where extended approximation of tissues under stress is required.

WARNINGS The safety and effectiveness of VICRYL (polyglactin 910) suture in neural tissue, and in cardiovascular surgery have not been established.

Under certain circumstances, notably orthopedic procedures, immobilization by external support may be employed at the discretion of the surgeon.

Do not resterilize

PRECAUTIONS VICRYL suture knots must be properly placed to be secure. Place the first throw in precise position for the final knot, using a double loop; lie the second throw square, using horizontal tension; additional throws are advisable.

Skin and conjunctival sutures remaining in place longer than 7 days may cause localized irritation and should be removed as indicated.

Acceptable surgical practice must be followed with respect to drainage and closure of infected wounds.

ADVERSE REACTIONS Reactions reported in clinical trials which may have been suture related have been minimal. These include skin redness and induration, rare instances of hemorrhage, anastomotic leakage, wound separation in the eye, and abscesses

DOSAGE AND ADMINISTRATION Use as required per

HOW SUPPLIED VICRYL sutures are available sterile, as braided dyed (violet) and undyed (natural) strands in sizes 3 to 8-0, in a variety of lengths, with and without needles, and on LIGAPAK* ligating reels. VICRYL sutures, monofilament, dyed (violet) are available in sizes 9-0 and 10-0, in a variety of lengths with needles.

Also available in sizes 1 to 4-0 attached to CONTROL RELEASE* needles.

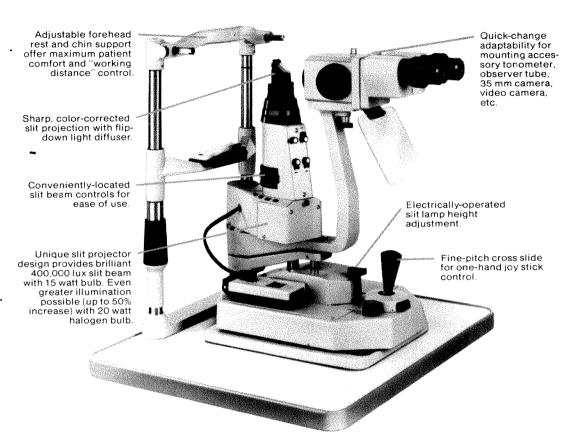
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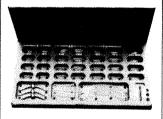
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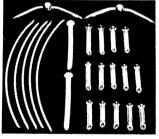
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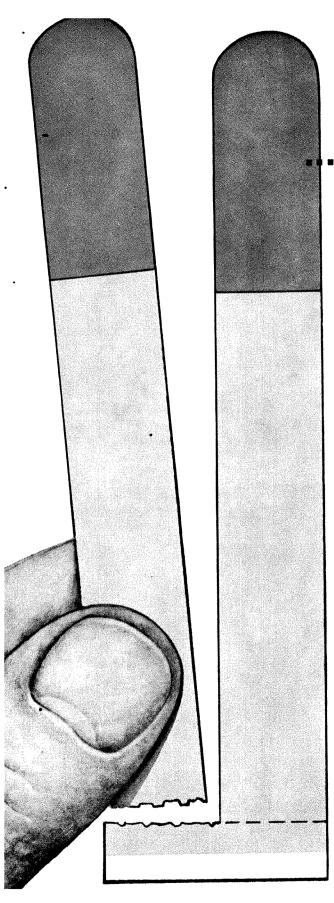
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- L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59. No. 5, May, 1965.
 - J.C. Mustarde', L.T. Jones M.D. and A. Callahan M.D., Ophthalmic Plastic Surgery-
- Up-To-Date, Aesculapius Publishing Company, 1970. L.T. Jones M.D. and J.L. Wobig M.D., Surgery of the Eyelids and Lacrimal System, Aesculapius Publishing Company, 1976

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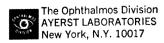


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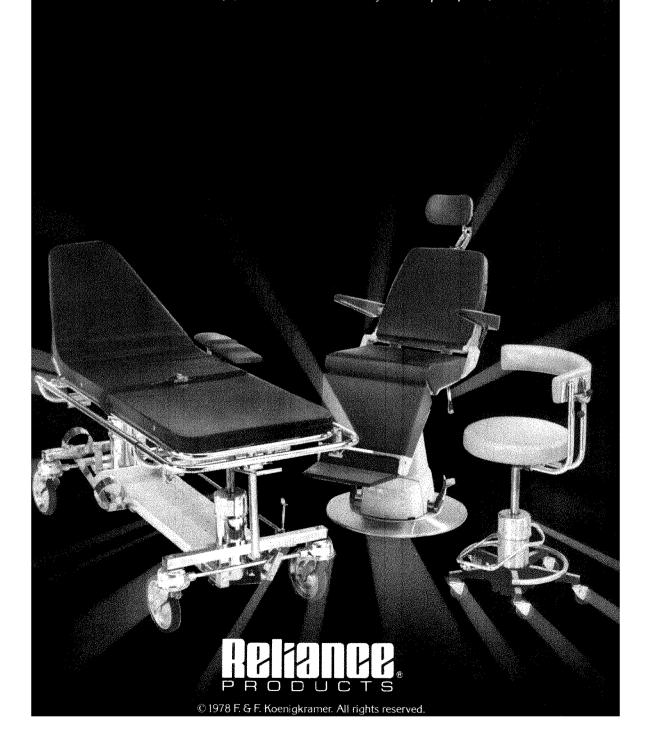
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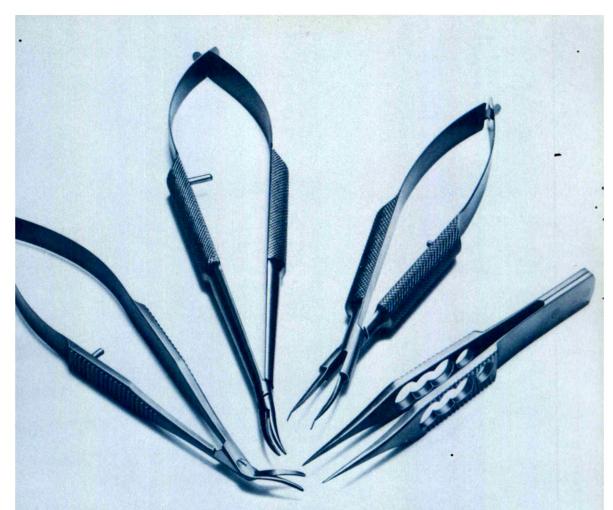


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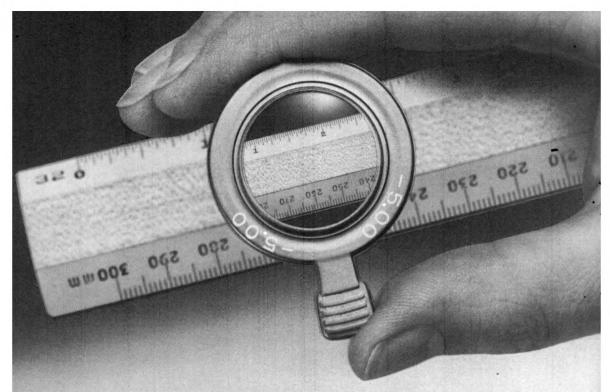
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Other major seminars will include Intraocular Lens/Cataract Surgery chaired by Dr. Richard Troutman, Cornea and External Diseases chaired by Dr. Herbert Kaufman, Contact Lens Update chaired by Dr. Antonio Gasset, Vitreous/Choroid/Retina chaired by Dr. Harvey Lincoff, and Cosmetic Surgery chaired by Dr. Pierre Guibor, all with panel discussions and question-and-answer sessions.

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PRECAUTIONS The solutions are incompatible with silver preparations. Ophthalmic ointments may retard corneal healing. Non-susceptible organisms including fungi may proliferate with the use of these preparations. Sulfonamides are inactivated by the para-aminobenzoic acid present in purulent exudates. Sulfonamide sensitivity reactions may occur. sensitivity reactions may occur.

June 1972

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INDICATIONS — Suspension Based on a review of this drug by the National Academy of Sciences — National Research Council and/or other information, FDA has classified the indi-

Council and/or other information. FDA has classified the indications as follows.
Possibly' effective: for the treatment of the following inflammatory and altergic conditions affecting the eyelids and anterior segment of the eye. EYELIDS altergic blephanitis, blephanitis associated with seborrheir dermatitis and other nonpurulent forms of conjunctivitis including those associated with hay fever and conjunctivitis due to physical agents such as foreign bodies, chemicals racids, alkalies) and other intrants. CORNEA, SCLERA, IRIS, AND UVEA: interstitial, postoperative, and sclerosing keratitis; chemical and thermal burns of the cornea; corneal ulcer herpes zoster ophthalmicus, phylctenular keraticonjunctivitis, corneal neovascularization; scleritis, episcleritis, acute, chronic, and traumatic indocyclitis. Final classification of the less-than-effective indications requires further investigation.

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conneal neovascularization: scleritis: episcieritis: acute chronic and traumatic iricocyclitis in deep-seated infections such as endophthalmitis, panophthalmitis and orbital cellulitis, or when systemic infection threatens specific oral (antibiotic, sulfonamide) therapy should be employed Local treatment may be used as adjunctive therapy.

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Ingredients

RECAUTIONS

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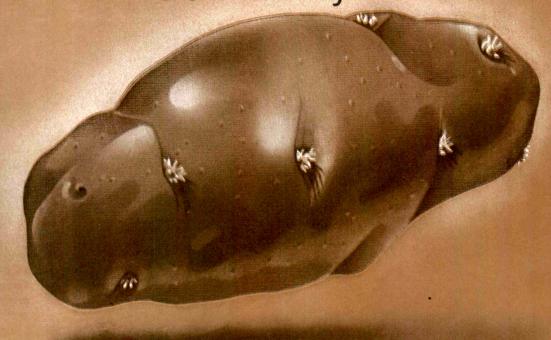
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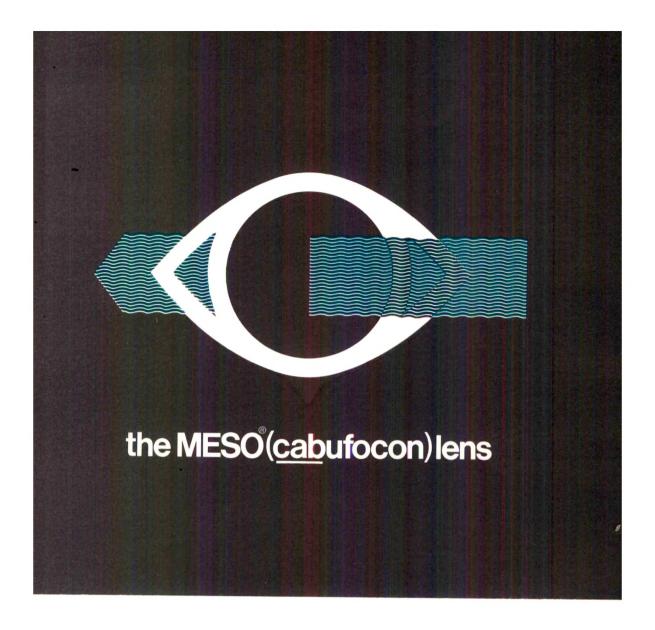
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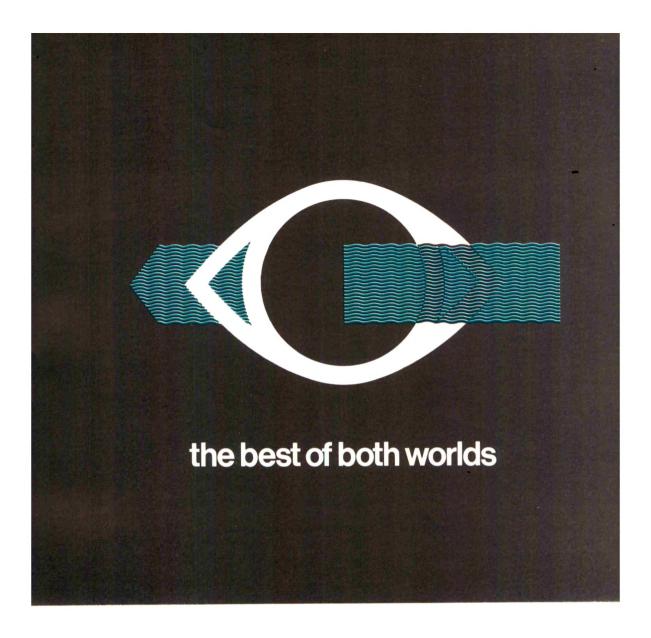
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- 2. Reduced Signs of Overwearing The investigators cited longer wearing periods. In 1,796 slit-lamp observations, a 1.2% incidence of corneal edema and spectacle blur was reported.
- **3. Flexibility and Ease of Fitting** Fitting The Meso Lens 0.25 diopter steeper or flatter, or with diameter differences of 0.1 or 0.2 mm, will not cause any significant change in comfort or acuity.

Parameters which affect tear exchange, such as apical clearance and intermediate bearing, are not critical. This increased latitude, particularly important in fitting astigmatic corneas, can be attributed to



the characteristics of the <u>cab</u>ufocon material as well as to the unique optical design of The Meso Lens.

4. Simplified Lens Care A three-solution maintenance system utilizing Preflex®, Normol®, and Flexsol® for cleaning, rinsing and disinfecting, is recommended for The Meso Lens.

Virtually no solution-related problems, such as discoloration, binding of preservatives or drug sensitivity, have been reported with The Meso Lens.

NOTE: Thermal disinfection or the use of hard lens care products is contraindicated.

5. Crisp Visual Acuity In a group of 773 eyes fit with The Meso Lens 97.3% tested 20/25 - or better than the best possible vision attained with spectacles in the same series.

Danker and Wohlk's exclusive molding process is utilized in the precision manufacture of The Meso Lens. The result: Uniformly good edges, high reproducibility and a smooth finish, which will resist accumulation of surface deposits.

The Meso Lens is available in a wide range of base curves and diameters, 6.0 to 9.0 mm and 6.0 to 12 mm respectively. The Meso Lens deserves a trial with new patients and wearers presenting problems with PMMA or HEMA lenses.

See package insert and professional fitting guide for complete information. For further details, call toll-free 800-645-1388 or write:

DANKER & WOHLK, INC.

635 Nassau Road, Uniondale, N.Y. 11553



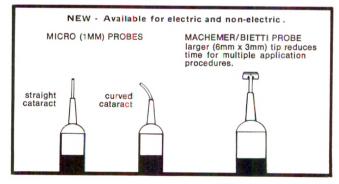
For the ophthalmologist (and everyone else), Keeler introduces a cryo system that makes the rounds . . . the new ACU22 cryosurgery unit.

Non-electric for instant freeze and defrost, the new ACU22 provides a variety of important, innovative features. And, using specially-designed Keeler/Amoils probes, the ACU22 performs as a multi-purpose cryosurgery/cryotherapy system, with applications for a broad range of medical specialties.

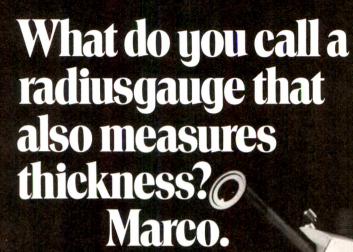
All ACU22 ophthalmic probes can also be used with the Keeler/Amoils ACU20 non-electric ophthalmic cryo unit (available at a slightly lower cost). But, for a cryosurgery system that's at home in any O.R., call or write for details on the new ACU22.

- Exhaust vent on back of console guides coolant gases (CO₂ or N₂O) out of the O.R. (via tubing).
- Pressure regulator knob on console makes remote adjustment on gas cylinders unnecessary.
- Outlet for external thermocouple, which monitors temperature when inserted in adjacent tissue.
- Switch converts probe-tip temperature meter into surrounding-tissue temperature meter.

APPLICATIONS: 19 different probes for use in ophthalmology, ENT, gynecology, proctology, dermatology, oral/dental and general surgical procedures.







Most radiusgauges measure only contact lens curvature. Now, with Marco's new external reading model, you can measure both radius and thickness on a single instrument. You get the functions of two instruments for far less than you would expect to pay for the standard radiusgauge.

In addition to the convenience of dual function, the sensitivity of both is greatly increased through the large external dial gauge and the 100x magnification. Critical focusing is also enhanced by a coaxial focusing knob which allows both coarse and fine adjustment. And to suit individual preferences, the instrument is available in both monocular and binocular models.

Marco wraps all these advantages in a compact instrument incorporating the transformer in the base of the unit.

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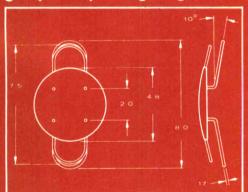
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ADSORBOCARPINE® pilocarpine HCl in Adsorbobase®

Description: Adsorbocarpine Ophthair Solutions are sterile, buffered, stabilized solutions of pilocarpine HCI (U.S.P.) in Adsorbobase — (a combination of high lecular weight, water soluble polymers a povidone). Adsorbocarpine is available in three strengths: 1%, 2% and 4%. All strengths, in addition to the respectiv amount of pilocarpine HCI, contain:

Vehicle: Adsorbobase and Hydroxyethylcellulose

Preservatives: Benzalkonium

Chloride0.0 Edetate Disodium ...0.1

Indications: For the control of intraocul pressure; as a miotic in the treatment of chronic, simple, open-angle glaucoma; to counteract the effect of cycloplegics. The choice of strength should be determined the severity of the condition and the resport the patient. To relieve tension in the trament of acute glaucoma Adsorbocarpine may be used alone — prior to emergency surgery — or in combination with other miotics or carbonic anhydrase inhibitors

Administration: The choice of concentrat and the frequency of instillation should be determined by the severity of the conditional the response of the patient.

Supplied: In 15 cc. sterile dropper vials — 2%, and 4% strengths.

Contraindications: The use of Adsorboca pine is contraindicated where pupiliary c striction is undesirable or hazardous such in acute iritis. Miotics should be used with caution in hypertension.

Precautions: Avoid overdosage.

Adverse Reactions: There are no known seffects except slight ciliary spasm with restant temporary reduction in visual acuity. Sensitivity is infrequently observed. Contallergy or mucosal sensitization may deveafter prolonged use. If signs of sensitivity velop during treatment, or if irritation persor increases, the patient should be advise discontinue use and consult the prescribil physician.

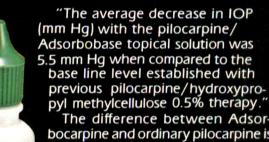
Caution: Federal (U.S.A.) law prohibits d pensing without prescription.

lough cases of primary, open-angle glaucoma may be controlled with Adsorbocarpine.



leavily pigmented, or brown, eyes are ifficult to control with pilocarpine. Yet Sherman's study with 26 heavily igmented eyes, Adsorbocarpine® uickly brought IOP under control in hese tough cases.

Following prolonged treatment vith a pilocarpine/hydroxypropyl nethylcellulose solution "20 of the 6 eyes (13 patients) were judged to xhibit uncontrolled IOP (above 1 mm Hg)... In every instance, the yes treated with the pilocarpine/dsorbobase solution [Adsorbocarine] were brought under control all but one patient ... this reduction in pressure was accomplished to a greatly reduced pilocarpine oncentration...



The difference between Adsorbocarpine and ordinary pilocarpine is Adsorbobase[®], a patented vehicle of high molecular weight, water soluble polymers which adsorbs to the cornea to enhance corneal penetration and availability of pilocarpine.

For your tough cases . . . recommend Adsorbocarpine.

See full prescribing information on facing page.

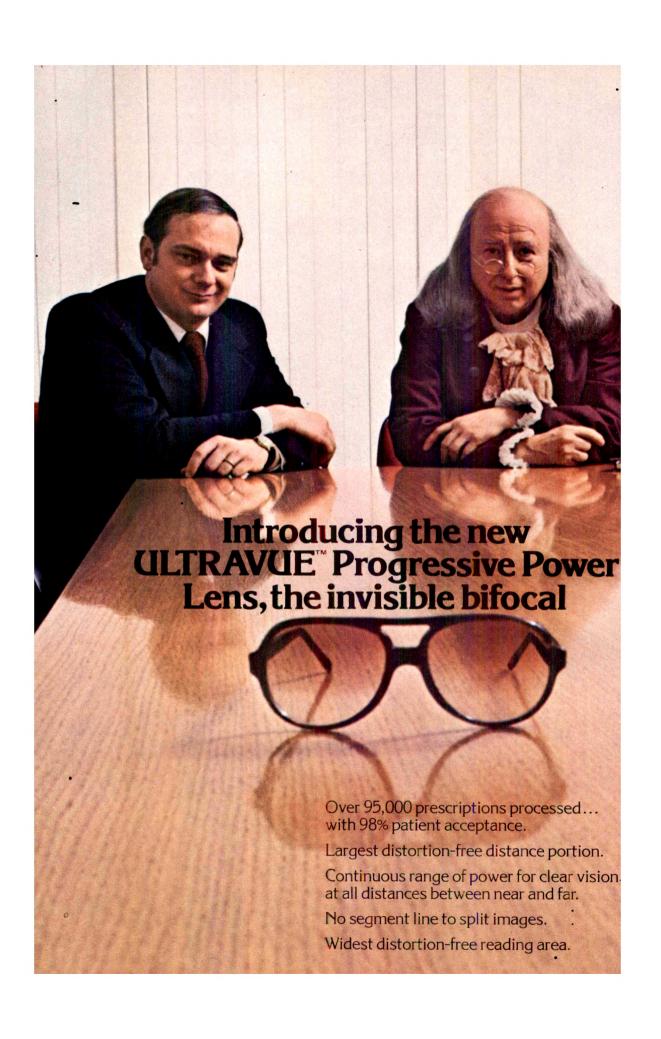
ADSORBOCARPINE®... pilocarpine HCI in Adsorbobase®...1%, 2% and 4%.



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Ophthalmic Products Division Central Industrial Park, Washington, D. C. 20027

1. Sherman, Spencer E.: Clinical Comparison of Pilocarpine Preparations in Heavily Pigmented Eyes: An Evaluation of the Influence of





..and the people who made it possible.

John Winthrop, Ph.D., Lens Designer. "Perhaps the greatest challenge was improving a lens concept that not only would permit add power at all distances between near and far, but more importantly provide an aberration-free distance portion, a wide aberration-free reading portion and a short continuous progressive addition corridor for intermediate viewing. I feel strongly that presbyopes, who might resist wearing bifocals, will wear ULTRAVUE Lenses because they have no discontinuity in power and no dividing lines between the lens portions for near and far."

Bernie Grolman, M.S., D.O.S., Fitting System Designer. "It's another case of necessity being the mother of invention. The ULTRAVUE Lens with its sophisticated optics requires precise fitting. A new instrument was developed that determines the critical measurements. The Grolman Fitting System, which is simple to use, is an integral part of a total program aimed at ensuring patient satisfaction."

Robert Platt, O.D., Clinical Trial Supervisor. "In a sense, the ULTRAVUE Lens was a natural for AOLITE® Lens material. Its lightweight, large blank size and wide prescription range makes the cosmetic appeal of the lens unquestionable. And there is no reason why presbyopes should be denied what single vision wearers have come to expect. Let's face it, cosmetic appeal is important today. But optical quality shouldn't be sacrificed."

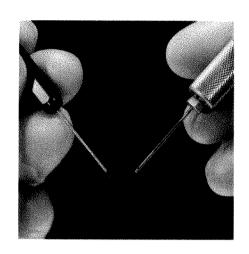
John Hancock, Product Manager. "Any innovation in optics carries responsibilities. The patient, the ophthalmologist, the dispenser—each has to know what it's all about, why it's better, why it was developed. And we have a responsibility to do our utmost to make sure the lens is fitted correctly. That's why we have trained over 22,000 people to successfully fit and fabricate the ULTRAVUE Lens in our certification program for dispensers and laboratories."

Dr. Benjamin Franklin, Bifocal Inventor. The only one involved in the development of the ULTRAVUE Lens who is not associated with American Optical. Dr. Franklin was unavailable for comment.

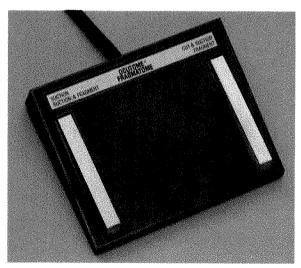
The new ULTRAVUE Progressive Power Lens...the revolutionary new lens Ben Franklin would be proud to recommend.

Optical

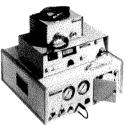
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The switch to the Total System.



Now, for total anterior segment surgery... Introducing Berkeley's superb, new ultrasonic fragmentor, the Fragmatome™ unit, for lens extraction. Together with the Ocutome® unit. In the only complete system that works with one, single footswitch.



A single footswitch....

Berkeley's Ocutome/Fragmatome System is the only system for total anterior segment surgery and posterior vitrectomy that works with a single footswitch like you see here.

But the exclusive features of our new system go well beyond our footswitch.

Ocutome/Fragmatome is the only complete system for total anterior segment surgery. including such procedures as lens removal, capsulectomy, iridectomy, pupillary reshaping, and removal of prolapsed vitreous.

The Ocutome/Fragmatome System

The Ocutome/Fragmatome System is the most miniaturized intraocular instrument available.

The system offers superior surgeon control....

You directly control suction, cutting rate, port opening size and ultrasonic power output.

And superior safety...

The Ocutome unit is a pneumatic system. So there are no electrical connections or shock hazards to the patient.

The Ocutome/Fragmatome System uses only tiny incisions for all 20-gauge pics. probes, needles and accessories. Smaller incisions minimize trauma and are easier to close water-tight.

Guillotine cutting action prevents spooling and pulling tissue. And provides safer cutting near the retina and safer reshaping of the iris.

Our automatic suction system is pressure-controlled—not flow-controlled—for a continuous level of suction for both tissue or lens removal.

And, a single cable connects both units to a single footswitch—for superior direct control in cutting and suction.

A few more special features.... Your choice of an infusion sleeve or modular infusion to automatically control intraocular pressure.

The Fragmatome unit offers a special wattmeter, an elapsed time meter and fine tuning controls.

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What's more, the system is economically priced and offers you more features and advantages than any other system.

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To help you put the Ocutome/Fragmatome System to best use. Berkeley's Educational Division has developed a nationwide, ongoing program of seminars in intraocular surgery—conducted by the leaders in the field.

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Upcoming workshops in intraocular surgery are planned for these dates at the locations listed here.

Call or write us direct for details and further information.

February 3. 4	San Jose, Ca.
March 17, 18	San Antonio, Tex.
April 21, 22	San Francisco, Ca.
April 28, 29	San Francisco, Ca.
Early May	Chicago, Ill.
Late May	New York, N.Y.
Mid June	Minneapolis, Minn.
Mid July	Cleveland, Ohio
Late August	Durham, N.C.
September 16, 17	Los Angeles, Ca.

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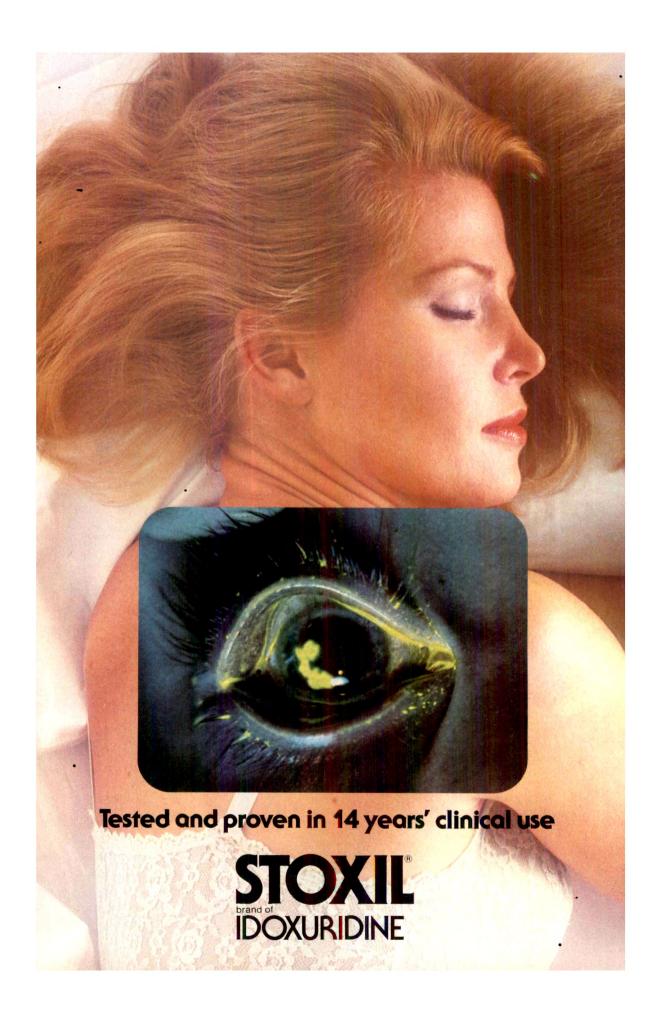
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Ointment 0.5% Solution 0.1%

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- Epithelial or Stromal Lesions

*Based on manufacturer's suggested list prices. Check your local pharmacy for actual patient cost.



Before prescribing, see complete prescribing information in SK&F literature or <u>PDR</u>. The following is a brief summary.

Indication: Herpes simplex keratitis (topical use only).

Contraindications: Known or suspected hypersensitivity to any of the components.

Warning: Administer with caution in pregnancy or women of childbearing potential. Malformations were reported in one study in rabbits when idoxuridine was instilled in the eyes of the dams; a subsequent, more detailed study in rabbits showed no such effects, even at substantially higher dosages.

Precautions: If there is no response in epithelial infections after 7 or 8 days, other therapy should be considered. Recommended frequency and duration of administration should not be exceeded. Not effective in corneal inflammations if herpes simplex is not present. Boric acid should not be used concomitantly. To insure stability is maintained, the solution should not be mixed with other medications.

Adverse reactions: Occasionally, irritation, pain, pruritus, inflammation, or edema of the eye or lid; rarely, allergic reactions have been reported. Photophobia has occurred. Occasionally, small punctate defects (which may be a manifestation of the infection), corneal clouding, and stippling of the corneal epithelium have been observed.

Supplied: 0.1% Ophthalmic Solution (1 mg./ml.) in 15 ml. bottles with dropper: 0.5% Ophthalmic Ointment (5 mg./gram) in 4 gram tubes.

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Department of Ophthalmology St. Vincent's Hospital and Medical Center of New York

ANNUAL CLINICAL UPDATE

Wednesday, Thursday & Friday

Robert A. D'Amico, M.D. Director, Department of Ophthalmology



November 1, 2 & 3, 1978

G.	Peter	Halberg,	M.D.
M	leeting	Coordin	ator

Wednesday, November 1

GLAUCOMA and CONTACT LENS UPDATE

G. Peter Halberg, M.D., Chairman

Thursday, November 2

CORNEA UPDATE

Robert A. D'Amico, M.D., Chairman

Friday, November 3

RETINA UPDATE

An illustrious faculty will be presenting the latest developments in the Cornea, Retina, Glaucoma and Contact Lens fields

REGISTRATION (Clinical Update) - \$240.00, includes luncheon each day and a social function \$140.00 for Residents

Saturday, November 4

SOFT CONTACT LENS UPDATE

This intensive, practical "Hands On" course will be under the direction of G. Peter Halberg, M.D., with the cooperation of a distinguished faculty and the major soft contact lens manufacturers.

REGISTRATION (Soft Lens Update) - \$140.00, includes luncheon and a social function

For registration or information for either or both meetings, contact:

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Faculty

Claes H. Dohlman, M.D., Boston, Massachusetts Stuart I. Brown, M.D., Pittsburgh, Pennsylvania Peter R. Laibson, M.D., Philadelphia, Pennsylvania Richard H. Keates, M.D., Columbus, Ohio

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Tuition is \$800.00. For further information and application forms, please write: Richard S. Ruiz, M.D. Chairman, Department of Ophthalmology, Hermann Eye Center, Hermann Hospital, 1203 Ross Sterling, Houston, TX 77030, Phone: (713) 797-1777.

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OPHTHALMIC

Solution-Sterile Ointment-Sterile

Each millior gram contains gentamicin sulfate equivalent to 3.0 mg gentamicin

DESCRIPTION Gentamicin sulfate is a water-soluble antibi-

offic of the ammoglycoside group active against a wide variety of pathogenic gram-negative and gram-positive bacteria GARAMYCIN Ophthalmic Solution is a sterile, aqueous solution buffered to approximately pH 7 for use in the eye Each mL contains gentamicin sulfate (equivalent to 3.0 mg gentamicin), disodium phosphate monosodium phosphate sodium chloride, and benzalkonium chloride as a preservative.

gentamiciti), disodium privapinati, monactive sodium chloride, and benzalkonium chloride as a preservative.

GARAMYCIN Ophthalmic Ointment is a sterile ointment, each gram containing gentamicin sulfate (equivalent to 3.0 mg gentamicin) in a bland base of white petrolatum, with methylparaben and propylparaben as preservatives.

ACTIONS—The gram-positive bacteria against which gentamicin sulfate is active include coagulase-positive and coagulase-negative staphylococci, including certain strains that are resistant to penicillin. Group A beta-hemolytic and non-hemolytic streptococci, and Diplococcus pneumonae. The gram-negative bacteria against which gentamicin sulfate is active include certain strains of Pseudomonas aerugmosa indole-positive and indole-negative Proteos species Eschericha coh Klebsiella preumoniae (Friedlander's bacillus). Haemophilus influenzae and Haemophilus aegyptius (Koch-Weeks bacillus). Aerobacter aerogenes Morasella lacunata (diplobacillus) of Morax-Axenteld), and Neisseria sepcies including Neisseria gonorrhoeae. Although significant resistant organisms have not been isolated from patients treated with gentamicin at the present time, this may occur in the future as resistance has been produced with difficulty in vitro by repeated exposures.

by repeated exposures INDICATIONS GARAMYCIN Ophthalmic Solution and Ointment are indicated in the topical treatment of intections of the external eye and its adnexa caused by susceptible bacteria Such infections embrace conjunctivitis, keratitis and keratoconjunctivitis, corneal ulcers, blephanitis and blephaniconjunctivitis, corneal ulcers, blephanitis and blephaniconjunctivitis acute meibomianitis, and dacryocystitis CONTRAINDICATIONS GARAMYCIN Ophthalmic Solution and Ointment are confraindicated in patients with known hypersensitivity to any of the components WARNINGS GARAMYCIN Ophthalmic Solution is not for injection. It should never be injected subconjunctivally, nor should it be directly introduced into the anterior chamber of the eye.

PRECAUTIONS Prolonged use of topical antibiotics may PRECAUTIONS Prolonged use of topical antibiotics may give rise to overgrowth of nonsusceptible organisms, such as fungi. Should this occur, or di-irritation or hypersensitivity to any component of the drug develops, discontinue use of the preparation and institute appropriate therapy. Ophthalmic continents may retard corneal healing.

ADVERSE REACTIONS Transient irritation has been reported with the use of GARAMYCIN Ophthalmic Solution.

Occasional burning or stinging may occur with the use of GARAMYCIN Ophthalmic Continent.

DOSAGE AND ADMINISTRATION GARAMYCIN Ophthalmic Solution: instill one or two drops into the affected eye every four hours in severe infections dosage may be increased to as much as two drops once every hour GARAMYCIN Ophthalmic Ointment: apply a small amount to the affected eye two to three times a day.

HOW SUPPLIED GARAMYCIN Ophthalmic Solution—
Sterile, 5-mt plastic dropper bottle, sterile, boxes of one and six Store away from heat
GARAMYCIN Ophthalmic Ointment—Sterile, 5-ounce

tube, boxes of one and six. Store away from heat

NOVEMBER 1973

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A CHOICE FOR INITIAL THER

acy-against conjunctivitis and other inections of external eye and adnexa due to a wide

rections of external eye and adnexa due to a wide range of susceptible pathogens.

Gram-negative: susceptible strains of: H. influenzae; E. coli; K. pneumoniae; M. lacunata; Enterobacter aerogenes (formerly Aerobacter); H. aegyptius and Neisseria sp., including N. gonorrhoeae.

Gram-positive: susceptible strains of: staphylococci and streptococci, including D. pneumoniae.

entamicin sulfate, U.S.P.

ml. or gram contains gentamicin sulfate equivalent) mg. gentamicin.

se see product information on facing page.

ue to susceptible pathogens

Problem pathogens: susceptible strains of: P. aeruginosa and Proteus sp. (indole-positive and -negative).

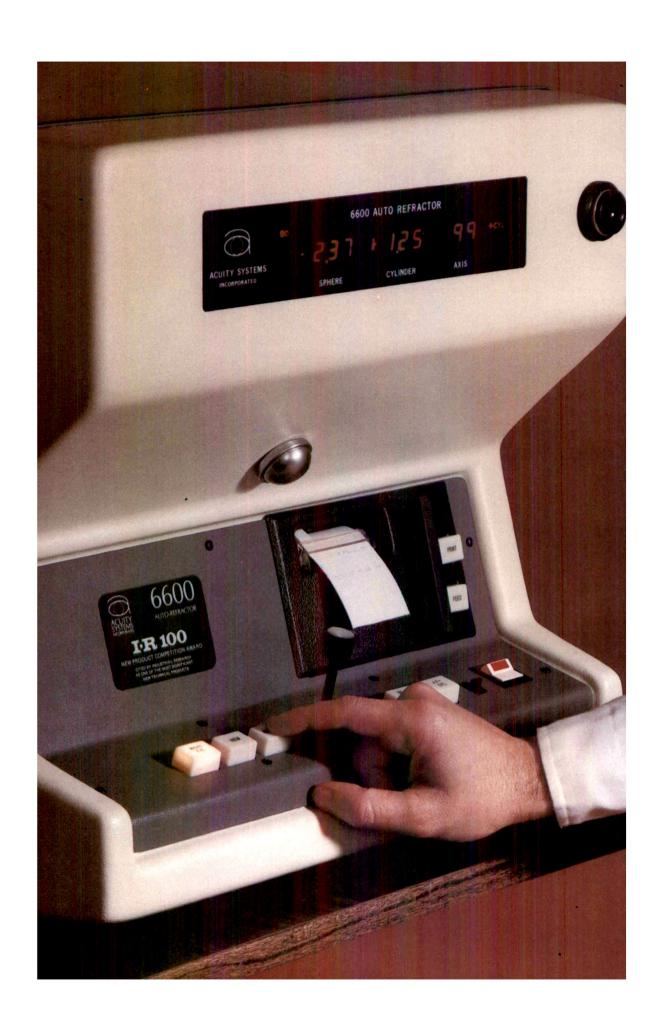
for gentle potency—generally avoids sensitivity reactions and irritation.

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broad spectrum activity against many gram-negative and gram-positive organisms

Solution-Sterile Ointment-Sterile

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Now's the time to automate the refractive part of your practice.

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As a professional, you want to give your patients the very best eye care possible. The Acuity Systems 6600 AUTO-REFRACTOR® adds the helping hand you need. Our new technology significantly improves your refractive capability over manual techniques. Poor responders and other difficult patients including aphakics, intraocular and contact ens patients, very young children, spectacle blur, scissors reflex, and early pathology patients are refracted faster and more accurately than is now numanly possible.

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Today's successful eye care practitioner faces nany challenges which, if met in an efficient way, present many opportunities.

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It's plain to see that NOW is the time to start providing superior eye care to your patients and to meet the challenges facing you and your practice. Send for the free booklet "Automated Refraction . . . it's Time". You'll learn why automated refraction is no longer a question of "IF", it's just a matter of answering, "When is it right for me?"

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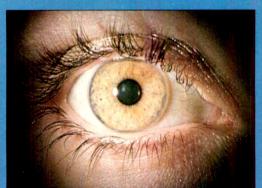
Autor if noze's the AUTO-REFRACTOR the free booklet, "Autor Name	Acuity Systems Incorporated 11413 Isaac Newton Sq. Reston, Virginia 22090 (703) 471-4700 The set to learn more about mated Refraction and see time to add the 6600 to my practice. Please send me mated Refraction it's Time"
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The Ferformance Story.

Soft contact lens performance that meets your exacting professional needs.

utstanding Visual Acuity

major benefit of Aquaflex® (tetrafilcon A) vdrophilic Contact Lenses is excellent visual uity. Clinical data revealed that 88% of the earers achieved 20/20 vision or better, with 3% attaining 20/25 or better.¹ Practitioners ported that Aquaflex lenses often corrected ore astigmatism and gave sharper vision an other soft lenses.¹ This quality of erformance is due to a unique combination sophisticated lens design, special lens aterial and the "Vault" system of fitting.

ptimum Centration

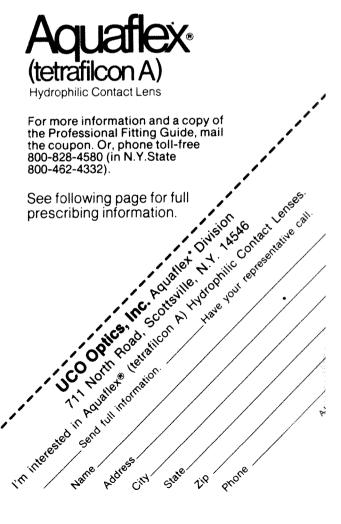
clinical study showed that over 96% of quaflex contact lens wearers achieved brimum centration.¹ By centering so well, quaflex lenses virtually eliminate the induced stigmatism and lessened visual acuity sulting from decentered lenses.² Precise entration helps in attaining a good fit with full brneal coverage and maximum visual acuity, hile permitting proper corneal respiration.

redictable Fit

o complicated graphs, charts and alculations, because the Aquaflex "Vault" ystem reduces fitting variables to just one... le lens "Vault". Only 5 Vaults are needed to fit quaflex lenses; and in clinical studies more an 90% of the patients were fitted with just 3 f these Vaults. The bicurve, lathe-cut lens onstruction, with spherical front and back urfaces, promotes reliable fit independent of ower; and the large posterior optical zone ives stability of vision with lens movement.

Additional Advantages

Other significant attributes of the Aquaflex® (tetrafilcon A) lens, contributing to practitioner success and patient acceptance, are: reliable over-refraction,²¹⁴ ease in handling, durability, excellent reproducibility² and exceptionally high standards of quality assurance, including 100% wet inspection. A study has shown that less than 5% of lenses shipped were returned because of questionable performance, discomfort, damage or defects.¹ The fitting procedure is simple and fast.⁴ You can fit from a small diagnostic set, or use a dispensing inventory. Lenses may be ordered by a toll-free phone call, and UCO Optics' service is quick and dependable. More and more practitioners are using Aquaflex lenses every day. Shouldn't you?



Data on file: UCO Optics, Inc., Scottsville, N.Y. 14546 Morrison, Robert J.: International Contact Lens Clinic, ummer 1976

Gruber, Ellis & Gordon, Stanley: Contact Lens Forum, ebruary 1977

. Greenspoon, Morton K.: Contact Lens Forum, lovember 1977.

Aquaflex is a registered trademark of UCO Optics, Inc.



DESCRIPTION

ACUAFLEX* (tetraflicon A) Hydrophilic Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The lens material, tetraflicon A, is a hydrophilic random terpolymer of 2-hydroxyethylmetbacrylate. N-vinyl-2-pyrrolidone and methylmethacrylate. The polymer is a three-dimensional network of terpolymer chains joined by divinylbenzene cross links. It consists of 57.5% tetrafilcon A and 42.5% water by weight when fully hydrated in normal saline solution. Lenses have a nominal diameter of 13 mm.

its hydrated state the AQUAFLEX* Hydrophilic Contact Lens is soft and pliable ministrywrated state the AUUAPLEX? Hydrophilic Contact Lens is soft and pliable When dry, the lens becomes hard and britle. These states are completely reversible and a lens which has been permitted to dry out will recover all of its hydrated properties when placed in normal saline for a period of two hours. When placed on the human cornea the hydrated lens acts as a refracting medium to compensate spherical ametropias. The material has a refractive index of 1.43 and the lens has a visible light fransmittance greater than 97%.

INDICATIONS

AOUAFLEX* Hydrophilic Contact Lenses are indicated for the correction of vision in persons with non-diseased eyes who have spherical ametropias, corneal astig-matism of 2.50 diopters or less and/or refractive astigmatism of 2.00 diopters

CONTRAINDICATIONS

AQUAFLEX* Hydrophilic Contact Lenses are contraindicated in the presence of any of the following conditions: (1) Acute and subacute inflammation of the anterior segment of the eye; (2) Any eye disease which affects the cornea or conunctiva (3) Insufficiency of lactimat secretion: (4) Corneal hypoesthesia; (5) Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS

Medications and Eye Drops: AQUAFLEX* Lenses must be stored in an appro-Medications and Eye Drops: AQUAFLEX* Lenses must be stored in an appropriate solution when off the eyes, the type of solution being dependent on the system used for disinfection. When the lenses are disinfected with the thermal disinfection system, they may be stored only in BOILINSOAK* Solution (sterile buffered isotonic solution containing boric acid, sodium borate, sodium chloride 0.7%, preserved with Thimerosal (Lilly) 0.01% and edetate disodium 0.1%). When lenses are disinfected with the chemical disinfection system, they may be stored only in FLEXSOL* Disinfection and Storage Solution (sterile buffered isotonic solution of sodium chloride, sodium borate, boric acid, polyvinypyrroidone, polyoxyethylene and polyoxypropylene, preserved with Thimerosal (Lilly) 0.01%, and edetate disodium 0.1%). No ophthalmic solutions or medications, including hard contact lens solutions.

No ophthalmic solutions or medications, including hard contact lens solutions, can be used by AQUAFLEX* Lens weards prior to or while the lenses are in place on the eyes. Also, no solutions, including hard contact lens solutions, other than BOILnSOAK* Solution or FLEXSOI.* Solution may be used on AQUAFLEX*

BOIL SOAK' Solution or FLEAGUL Subminion may be called the seem of the year of of the eyes.

Since liquids and vapors may be absorbed by AQUAFLEX' Lenses, they should not be placed in the mouth for wetting, nor wiped with a cloth or tissue.

Abrasions and infections: If a lens becomes less comfortable than it was when first placed on the wearer's cornea, the lens should be removed immediately and inst praced on the water's cornea, the tens should be removed immediately and the water's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, utceration, irritation or infection is present, or any abnimal eye condition is observed concurrently with lens wear, the lens should be removed immediately and a physician consulted.

Wearing Restrictions: AOUAFLEX* (tetraflicon A) Hydrophilic Contact Lenses exhalted to the search the control and the search the search the control and the search the search the search the control and the search the sea

should not be worn while swimming, sleeping, or in the presence of irritating furnes

Visual Blurring: If visual blurring occurs, the lens must be removed until the condition subsides.

Lens Care Regimen: Patients must adhere to the recommended daily care procedures for AQUAFLEX. Hydrophilic Contact Lenses. Failure to follow this procedure may result in the development of serious ocular infections.

Storage: AOUAFLEX' Lenses may be stored *anly* in the appropriate storage solution BOIL:nSOAK' Solution or FLEXSOL' Solution depending on disinfecting methods used if left exposed to are, the lenses will dehydrate, become brittle and break readily. If a lens dehydrates, it should be soaked in either BOIL:nSOAK' Solution or FLEXSOL' Solution until it returns to a soft, supple state.

Cleaning and Disinfecting: AQUAFLEX' Lenses must be both cleaned and disinfected daily. Separate procedures and products are needed to clean and to disinfect. Two methods of disinfection, thermal or chemical, have been shown to be equally effective. The choice of disinfection system should be made in consultation with your eye care practitioner

with your eye care practitioner
Cleaning: Daily cleaning is necessary to remove mucus and other deposits which
may have accumulated on the lens surface. Each time the lenses are removed
from the wearer's eyes, both surfaces of the lenses must be cleaned using several
drops of PREFLEX. Cleaning Solution (sterile buffered isotonic aqueous solution
consisting of sodium phosphates, sodium chloride, tyloxapot, hydroxyethylcellulose and polynynyl alcohol with Thimmerosal (Lilly) 0.004% and declate disodium 0.2% added as preservatives). Lenses must be cleaned before they are
disinfected, as deposits on the lenses tend to harden and become more difficult to
remove after the lenses are disinfected.

Disinfecting: AQUAFLEX* Lenses may be disinfected with either a heat or chemical regimen. One method or the other must be selected, but not both. The user must not alternate between methods

Thermal Disinfection Method: AQUAFLEX* Lenses may be effectively disinfected after cleaning with PREFLEX*. Cleaning Solution with use of the AQUASEPT* Patient Unit and BOILnSOAK* Solution. Fresh BOILnSOAK* Solution must be used for daily storage of lenses or each time the lens is stored. The AQUASEPT* Patient Unit requires distilled water. The AQUAFLEX* Lens Storage Containers must be emptied and filled with fresh BOILnSOAK* Solution just prior to disinfecting the lenses.

Chemical DisIntection: Disinfection with PREFLEX* Cleaning Solution.
FLEXSOL* Solution and NORMOL* Rinsing Solution (sterile buffered isotonic aqueous solution of sodium chloride, sodium borate and bonc acid, preserved with Thirmerosal (Lilly) o 001%, edetate discolum 0 1% and chlorhexidine (0.05%) has also been shown to be an effective disinfection system for daily care of AQUAFLEX* Lenses AQUAFLEX* Lenses must be cleaned and rinsed daily (or after wearing) with PREFLEX* Cleaning Solution and NORMOL* Rinsing Solution. The AQUAFLEX* Lens Storage Containers must be emptited and refilled with fresh FLEXSOL* Solution each time the lens is stored. Fresh FLEXSOL* Solution must be used daily for storage and disinfection. WARNING DO NOT MIX CRIALTER-NATE THE DISINFECTION AND STORAGE SYSTEMS. FLEXSOL* SOLUTION SHOULD NOT BE USED WITH HEAT.

SHOULD NOT BE USED WITH HEAT. Hyglene: Before handling the lenses, hands must be washed, rinsed thoroughly and dned with a lint-free towel. Cosmetics, lotions, soaps, oils and hand creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Fluorescelin: Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescen is used, flush the eyes with normal saline solution and wait at least one hour before replacing the lenses. Too early replacement may allow the lenses to absorb residual fluorescein.

ADVERSE REACTIONS
Serious corneal damage may result from wearing lenses which may have soaked in hard contact lens solutions. Eye irritation may occur within a short time after puting on a hypertonic lens. Removal of the lens will relieve the irritation. Yery rarely a lens may adhere to an eye as a result of a patient sleeping with the lens on, or as a result of wearing a hypotonic lens. If a lens adheres for any reason, the patient may be instructed to apply a few drops of BOIL nSOAK* Solution (if using a thermal disinfection regimen) or DAPETTES* Lutricating Solution (fullered isotonic aqueous solution containing ADSORBOBASE* (polyviny)-pytrolidone with other water soluble polymers) with Thimerosal (Lilly) 0.004% and edetate disodium 0.1% added as preservatives) (if using a chemical disinfection regimen), and wait until the lens moves freely before removing it.

Clinical studies indicate that corneal edema as manifested by symptoms such as rainbows or halos around light or visual blurring may occur if lenses are worn continuously for too long a time. Removal of the lenses and a rest pend of at least one hour generally relieve these symptoms. If symptoms do not subside promptly, professional consultation shoulde be obtained.

Excessive tearing, unusual eye secretions and photophobia are not normal; if

Excessive tearing, unusual eye secretions and photophobia are not normal; if these symptoms occur, the patient should be examined to determine their cause.

DOSAGE AND ADMINISTRATION

Fitting: Conventional methods of fitting contact lenses do not apply to AQUAFLEX* (tetraficon A) Hydrophilic Contact Lanses. For a detailed description of the fitting technique, refer to the Professional Fitting Quide for AQUAFLEX* Hydrophilic Contact Lenses, copies of which are available from UCO Optics, Inc. Scottsville, New York 14546

Wearing Schedule: There may be a tendency for the patient to overwear the lenses initially. Therefore, the importance of adhering to the following initial daily wearing schedule should be stressed to the patient.

Day	(Hours)	(Hours)	(Hours)
1	4	2	4
2	4	2	4
3	5	2	5
4	6	2	5
5	7	2	5
6	7	1	6
7	8	1	7
8	8	1	8
9	9	1	8
10-14	10	1	balance of
15	all waking hours		waking hourst

tienses should never be worn 24 hours a day

Lens Care and Handling: Care must be taken on the initial visit to assure that the patient is supplied with an appropriate AOUAFLEX* Patient Care kit and fully understands all care and handling instructions for the lenses. As with any contact lens, regular recall visits are necessary to assure patient health and compliance with instructions.

How Supplied: Each lens is supplied sterile in a glass vial containing normal saline solution. The glass vial is marked with the vault number, dioptric power, and manufacturing identification number.

The AQUAFLEX* Patient Care Kit is required for lens cleaning, disinfection and storing of the lenses. The Kit may consist of either of the following:

nal Disinfection Regimen AQUAFLEX* Patient Care Kit

AQUASEPT* Patient Unit	Catalog No. A0101
AQUAFLEX* Lens Storage Container	Catalog No. A0201
PREFLEX* Cleaning Solution	Catalog No. A1201
BOILnSOAK* Solution	Catalog No. A1301
AQUAFLEX* Patient Instruction Book	Catalog No. X0102
Chemical Disinfection Regimen	
AOLIAFI FX Patient Care Kit	

AGOAFLEX Fallent Care Kit	
AQUAFLEX* Lens Storage Container	Caratog No A0202
PREFLEX* Cleaning Solution	Catalog No. A1201
NORMOL* Rinsing Solution	Catalog No. A1401
FLEXSOL* Disinfection and Storage Solution	Catalog No. A1501
ADAPETTES* Lubricating Solution	Catalog No. A1601
AQUAFLEX Patient Instruction Book	Catalog No. X0102

AQUAFLEX and AQUASEPT are registered trademarks of UCO Optics, Inc., PREFLEX NORMOLETIES and BOILINSOAK are registered trademarks of Burton, Parsney & Compa

CAUTION: Federal law prohibits dispensing without prescription

UCO Optics, Inc. Aquaflex Division Scottsville, New York 14546

X0302

Today's ration of tears

There's a good reason to remember that Albalon-A is the only decongestant-

antihistamine in a soothing, tear-like vehicle.
Years ago, Bennett¹ pointed out that lacrimal secretions total approximately one teaspoonful per day. It seems to make sense that any drops instilled into the eye should supplement this meager supply rather than reduce it in any way.

If your diagnosis calls for a decongestant-antihistamine combination, only Albalon-A assures your patient of medication delivered in a soothing, tear-like vehicle. Albalon-A is formulated in Liquifilm® (polyvinyl alcohol

Albalon-ATM

sterile ophthalmic solution

DESCRIPTION A sterile ophthalmic solution having the following composition: naphazoline HCI 0.05%, antazoline phosphate . . . 0.5%, with: Liquifilm® (polyvinyl alcohol) 1.4%: benzalkonium chloride .004%, edetate disodium; polyvinyl pyrrolidone: sodium chloride; sodium acetate, anhydrous acetic acid and/or sodium hydroxide if needed to adjust the pH. ACTION Albalon-A combines the effects of the antihistamine, antazoline, and the decongestant naphazoline.

INDICATIONS Based on a review of a related combination of drugs by the National Academy of Sciences - National Research Council and/or other information, FDA has classified the indications as follows: "Possibly effective: For relief of ocular irritation and/or congestion or for the treatment of allergic, inflammatory, or infectious ocular conditions. Final classification of the less-than-effective indication requires further investigation.

1.4%) which closely approximates human tears in many physical parameters. This means that Albalon-A cannot introduce the risk of iatrogenic dry eye as may be seen with simple aqueous solutions.

So for minor ocular irritation and congestion, of allergic or inflammatory etiology,* consider Albalon-A. Only Albalon-A provides rapid eye whitening and prompt, effective relief from itching and discomfort—while it helps patients preserve their teaspoonful of tears.

Albalon-A[™]

naphazoline HCI 0.05%, antazoline phosphate 0.5%

The *only* decongestant-antihistamine in a soothing, tear-like vehicle.

CONTRAINDICATIONS Hypersensitivity to one or more of the components of this preparation, WARNING Do not use in presence of narrow angle glaucoma. PRECAUTIONS This preparation should be used only with caution in the presence of hypertension, cardiac irregularities or hyperglycemia (diabetes). To prevent contaminating the dropper tip and solution, care should be taken not to touch the eyelids or surrounding area with the dropper tip of the bottle. Keep bottle tightly closed when not in use. Protect from light. ADVERSE REACTIONS The following adverse reactions may occur: Pupillary dilation, increase in intra-ocular pressure, systemic effects due to absorption (i.e. hypertension. cardiac irregularities, hyperglycemia) DOSAGE One or two drops instilled in each eye every 3 or 4 hours or less frequently, as required to relieve symptoms. HOW SUPPLIED 15 ml dropper-tip plastic dropper bottles. On prescription only.

1. Bennett, J.E. The management of total xerophthalmia, Arch Ophthal 81:667-682, 1969.

AllERCAN
Pharmaceuticals, Irvine, CA 92713



Eyedrops, not teardrops

Phospholine Iodide[®]

ECHOTHIOPHATE IODIDE FOR OPHTHALMIC SOLUTION)

for accommodative esotropia

The emotional impact of accommodative esotropia on a young child need not necessarily be followed by the trauma of surgery or the inconvenience and problems associated with wearing and caring for prescription lenses. The agent frequently used in the diagnosis of the condition is the same agent that can be used to correct the accommodative factor without inconvenience to the young patient.

In diagnosis...One drop of PHOSPHOLINE IODIDE 0,125% instilled daily in each eye prior to retiring, for two or three weeks, will help to determine if there is an accommodative basis for the esotropia.

In treatment...If there is a significant accommodative factor present, the continued use of PHOSPHOLINE IODIDE alone is often sufficient to correct the problem, as long as the drug is well tolerated. PHOSPHOLINE IODIDE acts by altering the accommodative convergence/accommodation relationship in a favorable way, so that near vision is obtained with less accommodative effort and fusion can frequently be reestablished. If corrective lenses are necessary, PHOSPHOLINE IODIDE may permit the use of single vision lenses instead of bifocals.

If surgery is necessary, postoperative use of PHOSPHOLINE IODIDE may help correct a residual deviation.

BRIEF SUMMARY

full prescribing information, see package circular.)

PHOSPHOLINE IODIDE*

(ECHOTHIOPHATE IODIDE FOR OPHTHALMIC SOLUTION)
PHOSPHOLINE IODIDE is a long-acting cholinesterase inhibfor topical use.

Indications: Glaucoma - Chronic open-angle glaucoma Subacute or chronic angle closure glaucoma after indectomy or where surgery is refused or contraindicated. Certain non-uvelic secondary types of glaucoma, especially glaucoma following cataract surgery.

Accommodative esotropia – Concomitant esotropias with a

- Accommodative esotropia Concomitant esotropias with a significant accommodative component.

 Contraindications: 1. Active uveat inflammation.

 2. Most cases of angle-closure glaucoma, due to the possibility of increasing angle block.

 3. Hypersensitivity to the active or inactive ingredients.

 Warnings: 1. Use in Pregnancy. Safe use of anticholinesterase medications during pregnancy has not been established, nor has the absence of adverse effects on the fetus or on the respiration of the proposition. tion of the neonate
- tion of the neonate
 2. Succinylcholine should be administered only with great
 caution, if at all, prior to or during general anesthesia to patients
 receiving anticholinesterase medication because of possible
 respiratory or cardiovascular collapse
 3. Caution should be observed in treating glaucoma with
 PHOSPHOLINE IODIDE in patients who are at the same time
 undergoing treatment with systemic anticholinesterase medications for myasthenia gravis, because of possible adverse additive
 effects.

- effects

 Precautions: 1 Gonioscopy is recommended prior to initiation of therapy

 2 Where there is a quiescent uveitis or a history of this condition, anticholinesterase therapy should be avoided or used cautiously because of the intense and persistent missis and citiary muscle contraction that may occur

 3 While systemic effects are infrequent, proper use of the drug requires digital compression of the nasolacimal ducts for a minute or two following instillation to minimize drainage into the nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation.

 4 Temporary discontinuance of medication is necessary if salivation uninary incontinence, discrete, a profuse sweating, muscle weakness, respiratory difficulties, or cardiac irregularities occur.
- 5. Patients receiving PHOSPHOLINE IODIDE who are ex 5 Patients receiving PHOSPHOLINE IODIDE who are exposed to carbamate or organophosphate type insecticides and pesticides (professional gardeners, farmers, workers in plants manufacturing or formulating such products, etc.) should be warned of the additive systemic effects possible from absorption of the pesticide through the respiratory tract or skin. During periods of exposure to such pesticides, the wearing of respiratory masks, and frequent washing and clothing changes may be advisable.
- advisable
 6. Anticholinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial astima, spastic gastrointestinal disturbances, peptic uber pronunced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects
 7. Anticholinesterase drugs should be employed prior to ophthalmic surgery only as a considered risk because of the possible occurrence of hyphema.
- 8. PHOSPHOLINE (ODIDE (echothiophate iodide) should be used with great caution, if at all, where there is a prior history of

- used with great caution, if at all, where there is a prior history of retinal detachment. Adverse Reactions: 1. Although the relationship, if any, of retinal detachment to the administration of PHOSPHOLINE IODIDE has not been established, retinal detachment has been reported in a few cases during the use of PHOSPHOLINE IODIDE. In adult patients without a previous history of this disorder.

 2. Stinging, burning, lacrimation, lid muscle twitching, conjunctival and citiary redness, browache, induced myopia with visual blurring may occur.

 3. Activation of latent initis or uveitis may occur.

 4. Inscysts may form, and if treatment is continued, may enlarge and obscure vision. This occurrence is more frequent in children. The cysts usually shrink upon discontinuance of the medication, reduction in strength of the drops or frequency of instillation. Rarely, they may rupture or break free into the aqueous. Regular examinations are advisable when the drug is being prescribed for the treatment of accommodative escitropia.

 5. Protonged use may cause conjunctival thickening, obstruction of nasolacrimal canals.
- 6. Lens opacities occurring in patients under treatment for claucoma with PHOSPHOLINE IODIDE have been reported.
- glaucoma with PHOSPHOLINE. IODIDE have been reported and similar changes have been produced experimentally in normal monkeys. Routine examinations should accompany clinical use of the drug.

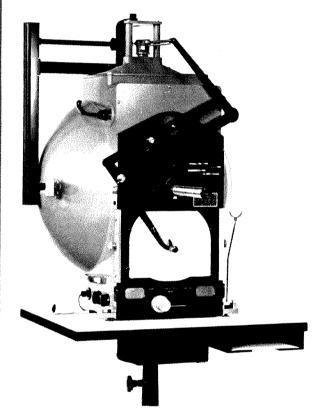
 7. Paradoxical increase in intraocular pressure may follow anticholinesterase instillation. This may be alleviated by prescribing a sympathominimetic mydinatic such as phenylephrine.

 Overdosage: Antidotes are atropine. 2 mp parenterally. PROTOPAM* CHLORIDE (praildoxime chloride). 25 mg per kg. pritravenously, artificial respiration should be given if necessary.

 How Supplied: Four potencies are available. 1.5 mg package for dispensing 0.03% solution. 3.0 mg package for 0.06% solution. 6.25 mg package for 0.125% solution. 12.5 mg package for 0.25% solution. Also contains potassium acetale (sodium hydroxide or acetic acid may have been incorporated to adjust pH during manufacturing), chorobutanol (chloral derivative), mannifol, boric acid and exsiciated sodium phosphate.

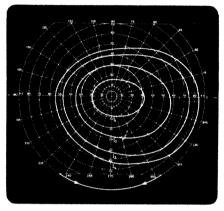


It's the Original! It's the Haag-Streit Perimeter 940



and now... delivery from stock

Originals have value in the healing arts as well as in the fine arts.
The GOLDMANN PERIMETER 940 by Haag-Streit is a prime example.



A product of the world's ophthalmological instrument leader, the Goldmann Perimeter provides high accuracy and economy of time.

The unit standardizes target size, luminosity of target and background and all other examination variables as well.

With its unique, patient-activated recording device, the Model 940 eliminates errors and simplifies target presentation control.

There are two original Goldmann models, the 940-K7 for kinetic perimetry and the 940-ST for static and kinetic perimetry.

If you are ready to make the lifetime investment that owning a true original represents, contact your Haag-Streit dealer. They will be glad to send full information on the Goldmann Perimeter and our many other fine products.

HAAG-STREIT

Haag-Streit Service, Inc.

Subsidiary of Haag-Streit AG., Berne, Switzerland

P.O. Box 127, 6 Industrial Park Waldwick, New Jersey 07463 (201) 445-1110

AN W Gener tion Of Retinal Cameras From Topcon,

Including two models with tilting facility.

Our new retinal cameras elevate fundus photography to a new level of ease and precision.

Here's how:

Simple and Accurate Horizontal Positioning

All TRC-FE series cameras feature a center of rotation at the pupil of the patient's eye. This allows the operator to easily

pivot the camera for peripheral photography. Newly-

designed base also facilitates stereo photography.

Tilting Mechanism

The TRC-FET and FET3 models also offer the user the advantage of a full 30° vertical tilt for greater control in positioning than can be achieved by altering patient fixation.

One-Handed Operation
Controls for alignment of the camera, height adjustment, and shutter release are all conveniently placed on the joystick for smooth, single-handed operation.

Many Other Important Benefits

All four TRC-FE series models provide an automatic film advance system, useful in fluorescein angiography. Our unique new Polaroid attachment (optional) permits taking of two side-by-side exposures on a single piece of Polaroid film. Standard features

include: Spectrotech Matched Interference Filter Set; macular fixation device; two camera bodies for color work and

fluorescein angiography; and an improved hinge mechanism on

fixation device which reduces the chance of breakage.

Best of all, the Topcon TRC-FE series of retinal cameras is available for a very moderate price. For a demonstration or more information, contact your local Topcon dealer or write to us.



A New World of Precision Optics

Topcon Instrument Corporation of America, 9 Keystone Place, Paramus, N.J. 07652

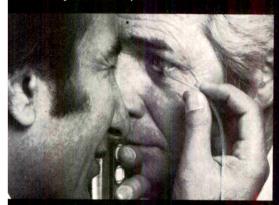
We I THE HIGH SHARE VILLIES, a new procedure that requires no assistance and is easier, faster and more accurate than conventional compression ophthalmodynamometry.

Self Positioning Cup

eliminates false readings associated with com-pression ophthalmodynamometry. Unlike compression instruments which must be held by an assistant absolutely perpendicular to the eye, the Digivac™ suction cup automatically assumes the correct attitude. Physician is free to look into the eye and watch for end points of the test without taking his eyes off the patient.

Jnique Hand Control

ttaches to ophthalmoscope or condensing lens, llowing physician to carry out test and record ressure without assistance. One-hand operation insures uninterrupted observation of diasolic and systolic end-points.

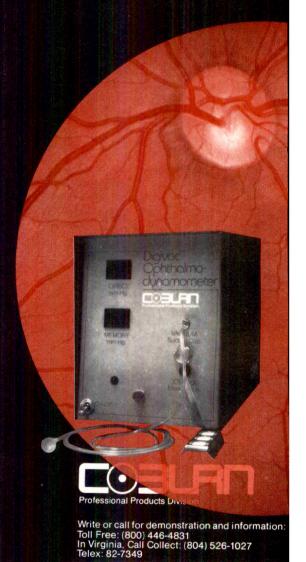


Digital Read-Out and Memory

ecords and stores diastolic pressures of carotid rtery as systolic pressure is being measured. Oth measurements are clearly displayed as ney are made, thus testing errors are virtually liminated. Readings are easily converted to the intraval cular pressure.

TM

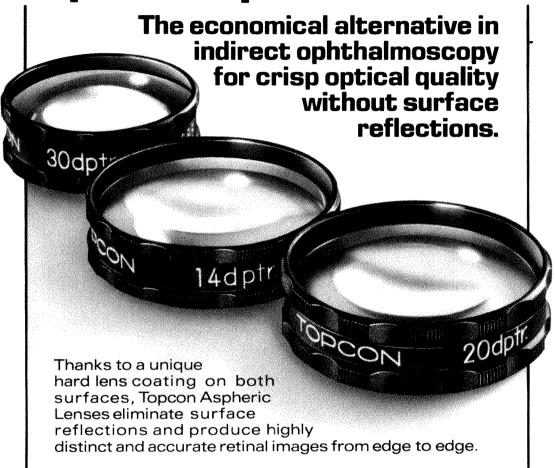
A fast, accurate and simple way o detect insufficiency in the nternal carotid artery system



Gentlemen: Please arrange a of the Digivac Op	demonstration ohthalmodynamor	meter
Name		
Address		
City	State•_	Zip
Telephone ().		
		410.01

Coburn Optical Industries, Inc. Professional Products Division P.O. Box 351 Petersburg, Virginia 23803

Topcon Aspheric Lenses



And as you might expect from Topcon, our new Aspheric Lenses give you more for less—superb Topcon optical quality combined with a significant price advantage.

Three powers are available: 14D, 20D, and 30D. Each lens is mounted in a lightweight aluminum ring and supplied with its own hard-shell case for easy carrying and safe storage.

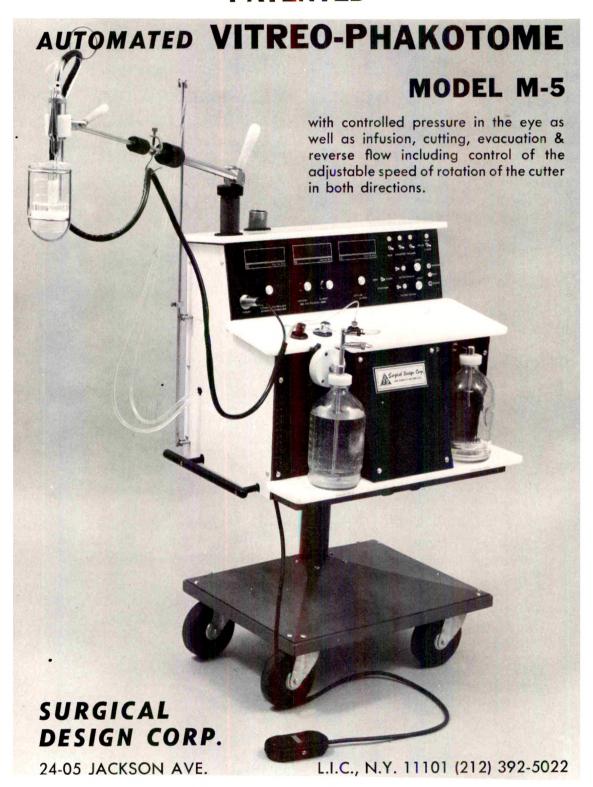
Consult your local dealer for our uncompromising economical alternative: the Topcon Aspheric Lens system.

TOPCON

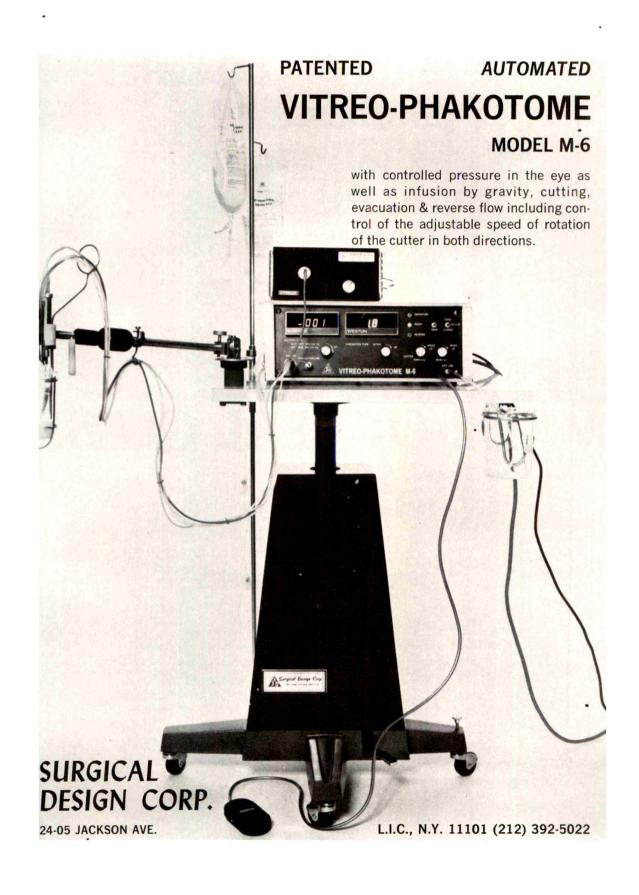
A New World of Precision Optics

Topcon Instrument Corp. of America, 9 Keystone Place, Paramus, New Jersey 07652

PATENTED



Vitreous Surgery: Arch. Ophthalmol. Vol. 93, August 1975



These days you can't take chances that what you dispense is <u>almost</u> right.

REAL GLASSES ARE MADE OF REAL GLASS

(and once they're made right they stay right.)

When glass spectacles are made to a correct prescription, they stay correct. Unlike plastic, they hold their shape won't bend or warp in their frames or wind up in a contour that's not *precisely* what you specified.

Glass is intrinsically harder and stronger than plastic. Lenses made from glass won't quickly scratch up or cloud up even accidental splashing with chemicals won't soften or dissolve them. And, specially strengthened glass lenses are impact resistant.

Let's be honest. Plastic is plastic. Real glasses are made from real glass.





10th ANNUAL ESTELLE DOHENY EYE FOUNDATION CONFERENCE

DIAGNOSTIC TECHNIQUES IN OPHTHALMOLOGY



Practical Aspects of Interpretation and Therapy • December 14, 15, 16: 1978
University of Southern California Health Sciences Campus, Los Angeles, California

Ultrasonography—Electrophysiology—Orbital Diagnosis—Office Diagnostic Techniques—Fluorescein Angiography—Oncology/Pathology/Clinicopathologic Correlations

Thursday: Basics A & B Scan, Exam Techniques, Intraocular Diagnosis, Vitrectomy Evaluations, Pre-operative Cataract Evaluation, Intraocular Foreign Bodies, Retinal Physiology, ERG, EOG, VER, Clinical Applications of Electrophysiology, Color Vision Testing, Specular Microscopy, Tear Deficiency, Visual Acuity, Bright Flash

Friday: Radiological Techniques in Orbital Diagnosis, Computerized Tonography, Ultrasound & Doppler in Orbital Diagnosis, Laboratory Diagnosis of Thyroid Disease, Drugs in Ocular Diagnosis, Background, Basics & Perspectives in Fluorescein Angiography, Retinal Vascular Disease, Diabetes, Intraocular Tumors, Iris Angiography, Macular Diseases,

Saturday: Melanotic Epibulbar Lesions & Syndromes, Cornea and Conjunctival Epithelial Tumors, Cytology in Ocular Diagnosis, Iris Tumors, Lid Lesions, Intraocular Melanomas, Oncology and Ocular Melanomas, Lymphoid Tumors, Electron Microscopy, Participating workshops in Ultrasound, Pathology, Microbiology and Electrophysiology.

The Estelle Doheny Memorial Lecture Lorenz E. Zimmerman, M.D. The A. Ray and Wendell C. Irvine Memorial Lecture William Hoyt, M.D.

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> REFERENCE NOTES: 1. Leiboweitz, H. M. and Kupferman, A. Bioavailability and therapeutic effectiveness of topically administered controosternids. Trans Am Acad Ophthalmol Otolaryngol 79 (1): op 78-op 88, 1975, 2, Ibid. 3, Ibid.



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THE ESSENTIAL IRIS ATROPHIES

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The first clear description of essential iris atrophy is generally credited to Harms¹ in 1903. Subsequently, nearly 200 cases have been reported in approximately 115 publications in the world. Because all of these reports have been of single cases or of a small series of cases, it has been difficult to develop a comprehensive understanding of this complex disorder. The purpose of this paper is to present the first large group of patients with the essential iris atrophies and to emphasize the clinical picture and course of the disease.

SUBJECTS AND METHODS

We studied 82 consecutive cases of essential iris atrophy from the private practice of Paul A. Chandler, M.D., and one of us (R.J.S.) and from the

Glaucoma Services of the Massachusetts Eye and Ear Infirmary and the Duke University Eye Center.

Patient selection was based on the definition of essential iris atrophy given by Chandler and Grant.² They describe three principal features of the disease: (1) distortion and atrophy of the iris; (2) corneal edema caused by dystrophy of the corneal endothelium; and (3) peripheral anterior synechiae of the iris to the cornea at Schwalbe's line or anterior to Schwalbe's line. Each patient in this study had at least two of these three features.

All of the patients were examined by Dr. Chandler or one of us. Fifty patients were observed for 1½ months to 36 years, with an average of 6.9 years. The remaining 32 patients were seen only on a single consultation visit.

RESULTS

General features—The age at which the patient first noted an abnormality or the age at which the diagnosis was made ranged from six to 58 years, with an average of 38.6 years. There was a 2:1 preponderance of women (56 to 26). All patients whose race was recorded were white.

From the Duke University Eye Center (Dr. Shields), Durham, North Carolina; Department of Ophthalmology (Dr. Campbell), Emory University, Atlanta, Georgia; and the Massachusetts Eye and Ear Infirmary (Dr. Simmons), Boston, Massachusetts. This study was supported in part by the Public Health Service research grant 2-ROI-EY00002 from the National Eye Institute and a grant from the National Society for the Prevention of Blindness, New York, New York.

Reprint requests to M. Bruce Shields, M.D., Duke University Eye Center, Durham, NC 27710.

Twenty-nine patients initially complained of visual disturbance; this complaint was most common. Patients usually described this as halos around lights or blurred vision or both. The visual disturbance was occasionally intermittent and was frequently more noticeable early in the morning. The second most common complaint was change in the appearance of the iris; it occurred in 25 patients. Sixteen patients described the change as a distorted pupil, but five described it as an extra pupil, and three, as a discoloration of the iris. The third most common initial complaint was pain, occurring in eight cases. However, pain was usually not noted until after visual disturbance or iris change had occurred. Six cases were discovered during an examination for an unrelated problem.

No additional ocular or systemic condition occurred frequently enough in these 82 patients to suggest a relationship to essential iris atrophy. The family history may have been contributory in three cases. In one, the mother was believed to have heterochromia. In the other two cases, one parent was said to have glaucoma. The glaucoma was bilateral in one case and was diagnosed at age 70 years in the other case. None of these relatives was available for examination.

Comeal abnormalities—We listed the corneal abnormalities in the eyes with essential iris atrophy (Table 1). An endothelial disturbance was specifically noted in 45 cases and was suspected in four others. This was often described as having a fine, beaten silver appearance (Fig. 1).

Twenty-two of the eyes with endothelial abnormality had associated edema of the stroma and epithelium. Nineteen additional eyes had stromal and epithelial edema, but no definite endothelial abnormality could be seen. Neither endothelial abnormality nor edema was reported in the remaining 14 case records.

TABLE 1
CORNEAL ABNORMALITIES
IN INVOLVED EYE ONLY

Abnormalities	No. of Cases
Endothelial abnormality	nadakan di hada di salu ada silisi d i dan pada karab dia pina anay yang da pay <u>dib</u>a diya
Definite	45
Questionable	4
Stromal/epithelial edema	
With endothelial abnormality	22
Without definite endothelial	
abnormality	19
No definite endothelial	
abnormality or edema	14
Other abnormalities	
Stromal scar	2
Recurrent erosion	1
Keratoconus	1
Band keratopathy	1

Iris abnormalities—The pupil was distorted in 58 cases. In 44 of these cases, the pupil was distorted in a single direction (Fig. 2); there was a random distribution regarding the quadrant toward which the



Fig. 1 (Shields, Campbell, and Simmons). Slitlamp view showing fine beaten silver appearance of corneal endothelial abnormality (arrows).



Fig. 2 (Shields, Campbell, and Simmons). Pupil is distorted in a single direction with ectropion uvea (EU). Iris stroma is thinned (T) in some areas and absent (A) with exposure of iris pigment epithelium in others.

pupil was distorted. In nine cases, the pupil was distorted in two opposing directions thus forming a spindle-shaped pupil (Fig. 3). Five pupils were only slightly irregular, and the rest appeared normal.

Ectropion uveae was present in 23

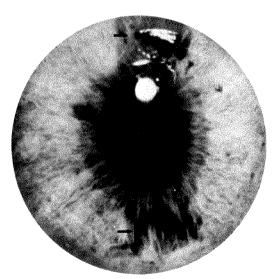


Fig. 3 (Shields, Campbell, and Simmons). Pupil is distorted in two opposing directions. Iris stroma is thinned or absent at both poles of the pupillary distortion (arrows).

cases (Fig. 2). In seven cases, the pupillary portion of the iris was pulled anteriorly away from the lens.

Atrophy of the iris stroma was reported in 58 cases. This was in a single area in 26 eyes, in multiple areas in 23, and was diffuse in nine eyes. These changes ranged from a mild thinning of superficial iris stroma (Figs. 2 and 3) to complete absence of the stroma, and underlying pigment epithelium was exposed (Figs. 2 and 3). Atrophy was specifically noted to be absent in eight cases, and in many cases, iris details were hard to see because of corneal clouding.

Twenty of the 58 eyes with stromal atrophy of the iris had associated perforating holes. In these cases, both the iris thinning and the holes appeared to be associated with a stretching of the iris (Fig. 4). Seven additional cases had holes without definite surrounding atrophy (Fig. 5). Fluorescein angiography in two of the latter cases showed the hole was surrounded by an ischemic zone, and leaking vessels were at the periphery of the ischemic area (Fig. 6).

The iris atrophy and holes were in the quadrant away from the direction of pu-

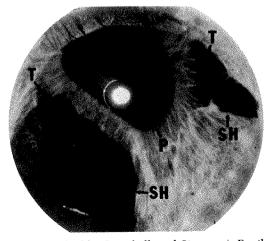


Fig. 4 (Shields, Campbell, and Simmons). Pupil (P) is distorted and iris is stretched with thinning (T) and stretch holes (SH).

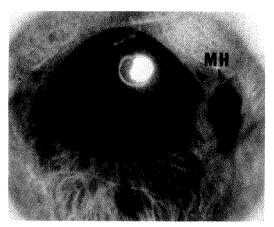


Fig. 5 (Shields, Campbell, and Simmons). Melting hole (MH) in eye with mild pupillary irregularity and ectropion uvea, but no significant stretching of iris

pillary distortion in 18 cases and in the quadrant toward which the pupil was distorted in 13 cases. The changes were too diffuse to correlate these two factors in the remaining cases.

Nine eyes had fine, pedunculated nodules on the iris. These appeared late in the course of the disease, progressed from yellow to dark brown, and eventually numbered 25 to several hundred (Fig. 7). Nodules were found in patients with mild, as well as severe, iris atrophy and

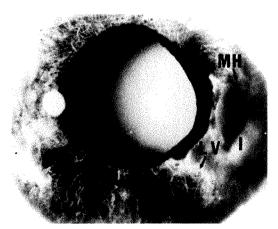


Fig. 6 (Shields, Campbell, and Simmons). Fluorescein angiogram of eye in Figure 5 shows area of ischemia (I) and leaking vessels (LV) around melting hole (MH).

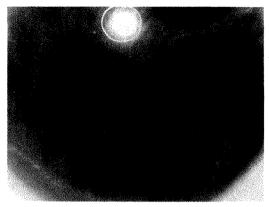


Fig. 7 (Shields, Campbell, and Simmons). Multiple fine pigmented iris nodules (IN) in eye with essential iris atrophy.

hole formation, and were occasionally associated with a flattening and loss of detail of the underlying iris stroma.

Anterior chamber angle abnormalities—Peripheral anterior synechiae were present in all but one of the 71 eyes in which gonioscopy was possible. The single eye in which no synechia was found had unilateral, fine endothelial abnormalities with mild stromal and epithelial edema of the cornea and mild stromal atrophy of the iris, but had a wide open, normal angle. The synechiae were single in 26 cases and multiple in 44. The extent of anterior chamber angle involvement with the synechiae is shown (Table 2).

With only one definite exception, the peripheral anterior synechiae extended to Schwalbe's line or anterior to Schwalbe's line in some area (Fig. 8). In six eyes, the synechiae bridged the angle in areas. Eight eyes had an accummulation of pigment on the synechia, especially in the most anterior portions. Three eyes had a yellow appearance at the interface of synechia and cornea.

Peripheral anterior synechiae were visible by slit-lamp examination on the peripheral cornea in 42 cases. Nineteen of these eyes had more than one area of visible synechiae. There appeared to be a

TABLE 2

EXTENT OF ANTERIOR CHAMBER
ANGLE INVOLVEMENT WITH
PERIPHERAL ANTERIOR SYNECHIAE

Percent of Angle Involved	No. of Cases
By single synechiae	
Less than 25	7
25-50	4
50-75	3
75 to total closure	12
By sum of multiple synechiae	
Less than 25	9
25-50	20
50-75	10
75 to total closure	5

random distribution regarding the quadrant in which the synechiae were seen. Occasionally, a yellow or white membranous structure was noted at the interface of synechia and corneal endothelium. One eye had an anterior synechia in midperipheral iris.

In 37 cases, the pupil was distorted toward the most prominent area of peripheral anterior synechia. In four other cases, the pupil was pulled into a spindle shape toward two main synechiae in opposite quadrants. In 28 eyes, the periph-

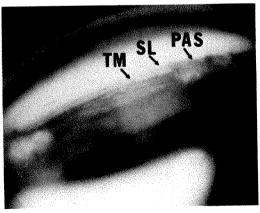


Fig. 8 (Shields, Campbell, and Simmons). Gonioscopic view showing peripheral anterior synechia (PAS) extending beyond trabecular meshwork (TM) to Schwalbe's line (SL).

eral anterior synechiae were so extensive that it was impossible to correlate them with the direction of pupillary distortion. In many of these cases, the pupil remained round or only slightly eccentric. In only one case was the pupil distorted away from the main area of peripheral anterior synechia.

Additional clinical abnormalities—The conjunctiva was hyperemic in six cases. All six were associated with increased intraocular pressure (IOP) or corneal edema, or both. Three eyes had an inflammatory reaction of the anterior chamber. The IOP in these eyes was 10, 20, and 30 mm Hg, and only one had corneal edema. Seventeen eyes had shallow anterior chambers. Nine eyes had lens opacities of some degree. Four of these had had filtering surgery.

The essential iris atrophy appeared to be unilateral in nearly all cases and equally distributed between right and left eyes. Several abnormalities were noted in the fellow eyes (Table 3). Four patients had one or more fine, peripheral anterior synechiae in the fellow eye. Two of these were associated with possible corneal abnor-

TABLE 3
STATUS OF FELLOW EYE

Abnormal Findings	No. of Cases
Peripheral anterior synechiae	gersygning dang pameng gryang ang ang arta banyilis dalah biraklabibat dalah
With corneal haze or dystrophy	2
With iris atrophy	1
With no other abnormality	1
Narrow angle	
In otherwise normal eye	12
With angle-closure glaucoma	1.
With plateau iris	1
Prominent iris processes	2
Open-angle glaucoma	1
Exfoliative glaucoma	1
Prominent Schwalbe's line	
and amblyopia	1
Ghost vessels in superior cornea	1
Congenital cataract and	
band keratopathy	1

malities and one was suspected to have mild iris atrophy. Fourteen of the fellow eyes had narrow angles. Table 3 lists the variety of other conditions that were noted in one or two cases.

Visual acuity—The distribution of visual acuities in the involved eyes is shown (Fig. 9). This represents the worst visual acuity in each case, either on the most recent visit or at the peak of corneal edema. Visual acuities ranged from 6/4.5 (20/15) to light perception, although 34 patients had a visual acuity of 6/9 (20/30) or better in their involved eye. The distribution of visual acuities was essentially the same regardless of whether the predominant feature was corneal edema, iris atrophy, or both.

Intraocular pressure—The distribution of the highest IOPs in the involved eyes is shown (Fig. 10). This ranged from 10 to 80 mm Hg, and averaged 29 mm Hg. In eves with corneal edema or endothelial abnormality, or both, the IOP ranged from 10 to 47 mm Hg, and averaged 23.8 mm Hg. In eyes with corneal edema, but no definite endothelial abnormality, the IOP ranged from 15 to 80 mm Hg, and averaged 33.3 mm Hg. In eyes with iris stromal atrophy and no iris holes, the IOPs ranged from 10 to 80 mm Hg, and averaged 27.2 mm Hg. In eyes with holes through the iris, the range was 14 to 62 mm Hg, with an average of 32.4 mm Hg.

Tonography was performed in 14 cases.

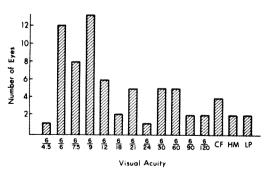


Fig. 9 (Shields, Campbell, and Simmons). Distribution of visual acuities in eyes with essential iris atrophy.

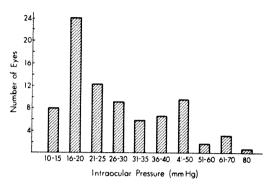


Fig. 10 (Shields, Campbell, and Simmons). Distribution of highest IOPs in eyes with essential iris atrophy.

The C-values ranged from 0.03 to 0.37 with an average of 0.19, and the PO/C ranged from 30 to 383, with an average of 119.8. Expressed as the C-value of the normal eye minus that of the involved fellow eye, the differences ranged from 0 to 0.25, with an average of 0.08. In no case was the C-value of the involved eye higher than that of the normal eye. The relationship between tonographic values and the extent of peripheral anterior synechial closure is shown (Fig. 11).

In three cases, serial tonographic tracings were obtained by W. M. Grant, M.D. He followed one of these cases for 15 years; during that time, the iridocorneal synechiae increased from 5 to 10 clock hours and the C-value decreased from 0.19 to 0.17, while that of the fellow eve remained at 0.22. The synechiae were observed to bridge the angle in some areas. In another case, observed for four years, the peripheral anterior synechia increased from 4 to 6 clock hours and the C-value decreased from 0.08 to 0.06. The third case, observed for one year, had a decrease in C-value of 0.33 to 0.13 with no apparent change in peripheral anterior svnechia.

Glaucomatous cupping was present in 22 of the 54 eyes with a visible optic nerve head. Glaucomatous field loss was present in 12 of the 31 cases in which this factor was studied.

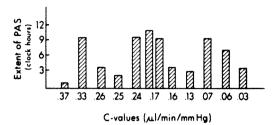


Fig. 11 (Shields, Campbell, and Simmons). Relationships between tonographic C-values and extent of peripheral anterior synechial closure; each bar represents one case.

The spectrum—The degree of corneal and iris abnormality varied greatly in these 82 cases. In one group of patients, the corneal abnormalities were predominant, with minimal iris changes. In another group, the iris abnormalities were predominant with perforating holes, and the corneal disturbances were variable. These two groups appeared to represent the extremes of a spectrum of varying corneal and iris abnormalities. The common feature throughout the entire spectrum was the presence of peripheral anterior synechiae, with the exception of the one case that has been previously mentioned. The distribution and overlapping of the corneal and iris abnormalities is shown (Table 4).

Course—An analysis of the cases followed for several years indicates that essential iris atrophy is a slowly progressive disease. Corneal abnormalities appear to be an early feature of the disease; they were noted on the initial visit in nearly all cases in which they occurred. Iris atrophy and holes were also noted on the initial visit in many cases, but in others they developed during the course of observation. Peripheral anterior synechiae were present on the initial visit in all cases except one and progressed gradually over the years in most cases. The circumferential extension of peripheral anterior synechiae was usually associated with a gradual increase in IOP, which often made the corneal edema worse. In many cases, the increased IOP went on to cause severe glaucomatous cupping and field loss.

Treatment—Sixty-three cases required treatment of the increased IOP. Twenty-five cases were adequately controlled by medical therapy. Filtering or other glaucoma surgery was performed in 31 cases. Twelve of these procedures were primarily for glaucoma, 12 for corneal edema, and seven for both. The types and results of these procedures are shown (Table 5). Two of the filtering procedures were combined with a wide excision of iris superiorly. This treatment controlled the pressure in one case, but resulted in severe corneal edema in both cases. Filtering surgery was recommended in five addi-

TABLE 4

OVERLAPPING OF CORNEAL AND IRIS ABNORMALITIES

		lities			
Corneal Abnormalities	No Apparent Abnormality	Pupil Distortion Only	Atrophy Without Hole	Holes	
No apparent		1	7	6	
abnormality Endothelial		-			
abnormality only	3	5	9	10	
Endothelial abnormality with corneal edema	1	4	11	6	
Edema without definite endothelial	1	2	11	5	
abnormality					

	TABLE 5	
RESULTS	OF GLAUCOMA	SURGERY

• Procedure	No. of Procedures*	Successful	IOP-Controlled, But Persistent Bullous Keratopathy	Failure	Not Stated
Trephine	19	7	3	5	4
Thermal					
sclerostomy	4	2	1		1
Posterior lip					_
sclerectomy	1		1		
Trabeculectomy	2		1	1	
Filtration (type			-	-	
not stated)	7	2	1	9	9
Cyclocryotherapy	2	-	1	2	2
Cyclodiathermy	1			ī	

^{*}More than one procedure in five cases.

tional cases, enucleation in one, and surgery was not recommended in one case because the optic nerve head was totally atrophied.

DISCUSSION

The general features of the patients in this study are consistent with most previous reports. In most cases, the condition first becomes noticeable in early or middle adult life. Although the average age at which the condition was first recognized in this study was 38.6 years, we do not know how long the condition may have been present before its recognition.

The present study suggests that the essential iris atrophies occur predominantly, if not exclusively, in white people. It is difficult to compare this observation with previous studies since most do not mention race. Women are affected more often than men. The 2:1 preponderance of women in this study is slightly lower than previous reports of up to 5:1.3

Almost all cases are unilateral. Four cases in the present study might represent bilateral involvement, since a few fine synechiae were present in the fellow eye. Two of these eyes had possible corneal

abnormalities and one other had mild iris atrophy. A similar bilateral case has been reported. Most of the reported cases that are definitely bilateral have additional features that are unusual in essential iris atrophy. In one report, a white man also had bilateral keratoconus. Another study reported four members of a black family of nine with bilateral progressive essential iris atrophy.

The intial complaints in this study illustrate the spectrum of manifestations in the essential iris atrophies. Visual disturbances, usually described as halos around lights or blurred vision, or both, were the initial complaint in 29 cases (35%). These are symptoms secondary to the corneal abnormalities. Iris changes constituted the initial complaint in 25 cases (30%). These changes were most often described as a distorted pupil, but occasionally as an extra pupil or a discoloration of the iris. Pain was the initial complaint in eight cases (9.8%). The pain resulted from extremely high IOPs or corneal edema, or both.

There does not appear to be any significant association of essential iris atrophy with other diseases. It may be significant that shallow anterior chambers were reported in 17 of the involved eyes and in 14 of the fellow eyes.

Familial occurrence is rare in the essential iris atrophies. Three patients in the present study had a family history that may have been contributory, but the relatives were unavailable for examination. A few familial cases have been reported, such as a white mother and daughter with unilateral involvement,⁷ and the previously mentioned black family with bilateral involvement in the father, a son, and two daughters.⁶

The clearest description of the characteristic corneal abnormality comes from Chandler's⁸ original article. He described "a fine hammered silver appearance similar to Fuchs' dystrophy but less coarse." This change involves the corneal endothelium, but the actual histologic alteration has not as yet been defined. Until the alteration in the corneal endothelium is better understood, we have elected to use the more nonspecific terms of endothelial disturbance or abnormality.

This characteristic corneal endothelial abnormality was specifically noted in 45 cases (55%) in the present study. In 22 of these cases, there was associated edema of the corneal stroma and epithelium. As Chandler⁸ noted, the corneal edema tended to occur at IOPs only slightly above normal. Nineteen additional cases (23%) also had corneal edema, without specific mention of any endothelial abnormality. In some of these cases, it is likely that the anterior corneal changes obscured the subtle posterior changes. However, in other cases it is possible that extreme increases in IOP could have caused the edema.

The iris changes are the most familiar feature of the essential iris atrophies. However, this aspect of the disorder varies greatly from patient to patient. In some cases, there was no detectable dis-

turbance of the iris or only minimal pupillary distortion or atrophy of the iris stroma. These mild iris changes often appeared nonprogressive and were nearly always associated with abnormalities of the corneal endothelium. In other cases, there was more extensive pupillary distortion and atrophy of the iris stroma. In 27 cases (33%), the atrophy progressed to formation of holes through the iris. The association of corneal abnormality with this form of iris change was more variable.

Iris nodules were noted in nine (11%) of the present cases. The details of this feature have been discussed in a previous report.9 Scheie and Yanoff 10 have reported similar cases, which differ from the essential iris atrophies only by the presence of a diffuse iris nevus or pigmented nodules. They consider this to be a separate disease and refer to it as the iris nevus syndrome. Since we have observed cases of essential iris atrophy for many years before the appearance of the nodules, we believe those cases with nodules represent a variation of the essential iris atrophies. This difference of interpretation has vet to be resolved.

The present study emphasizes the observation of Chandler and Grant² that the degree of corneal and iris change varies greatly from patient to patient. They describe two forms of essential iris atrophy: Chandler's syndrome and progressive essential iris atrophy. Although these two forms of the disease have features in common, such as peripheral anterior synechia and increased IOP, there are also significant differences.

In Chandler's syndrome, the dominant feature is an abnormality of the corneal endothelium with corneal edema at moderate IOP increases. The pupil remains round or only slightly distorted, iris atrophy is absent or mild, and holes do not develop. In progressive iris atrophy, the dominant feature is extensive atrophy of the iris and perforating holes. Abnormality of the corneal endothelium is not always observed, and the level of IOP at which corneal edema occurs is often higher than in Chandler's syndrome. Peripheral anterior synechiae are characteristically present in both forms of the disease.

The present study suggests that these two conditions represent two ends of a spectrum of the essential iris atrophies. Thirteen cases clearly satisfied the criteria for Chandler's syndrome in that definite corneal endothelial abnormality, with or without corneal edema, was present, and iris changes were mild or absent. Twenty-seven eyes satisfied the criteria for progressive essential iris atrophy by having iris holes with or without corneal abnormalities. However, the remaining 42 cases did not clearly satisfy the criteria for either form of the disease. Twenty cases had corneal abnormalities suggestive of Chandler's syndrome, but had more advanced iris atrophy without holes. Twenty-two cases had variable iris changes without holes. Fourteen of these 22 eyes had corneal edema with no definite endothelial abnormality. The other eight eyes had no apparent corneal abnormality.

Becker and Shaffer¹¹ stated that the natural course of the iris changes consists of

... thinning and eventual hole formation beginning in the midperiphery of the iris. The pupil is displaced away from this area. Soon peripheral anterior synechias begin to form on both the side of the holes and the opposite side toward which the pupil is displaced.

This description of the natural course of the disease implies that the iris changes precede the formation of peripheral anterior synechiae.

The present study suggests a different course. Since peripheral anterior synechiae were present on the initial visit in all cases but one, it is likely that they precede the iris changes. It appears that

the synechiae develop first in one area and that the pupil is then pulled in that direction. The iris on the opposite side is put on a stretch and eventually undergoes atrophy and, in some cases, hole formation. We propose to call these "stretch holes," in contrast to a less common type of hole, which occurred without associated thinning of the iris. The latter may occur in any quadrant, and fluorescein angiographic studies suggest that they occur in areas of iris ischemia. We propose to call these "melting holes."

Campbell, Shields, and Smith¹² have advanced the theory that a membrane, composed of a single layer of endothelial cells and a basement membrane, develops from a proliferation of the abnormal corneal endothelium and grows over the anterior chamber angle and the iris stroma. Subsequent contraction of this membrane produces the peripheral anterior synechia and pupillary distortion with subsequent iris thinning. The present study supports this theory in that the pupil is nearly always distorted toward the most prominant area of peripheral anterior synechia.

If the membrane theory proves to be correct, it may help to explain the unusual changes in the anterior chamber angle. Peripheral anterior synechiae were noted in all but one eye studied by gonioscopy. Most of these synechiae extended to Schwalbe's line or anterior to Schwalbe's line and in 42 cases (51%) they were visible on the peripheral cornea. The synechiae may have been pulled into this characteristic position by a membrane extending down from the cornea. The yellow or white membranous structure noted at the interface of cornea and synechiae in some cases may also be a manifestation of this membrane.

In the present study, it was difficult to consistently correlate the extent or progression of the peripheral anterior synechiae with tonometric or tonographic changes. One explanation for this discrepancy may have been the bridging that was observed in several cases. With bridging, aqueous may flow behind the synechiae in some areas, thus providing better outflow than the circumferential extent of synechiae would suggest. In other cases, the IOP increase seemed excessive in proportion to the apparent angle abnormalities. Obstruction of the angle by a membrane, which had not yet contracted to form synechiae, could account for this increase and might explain the one case in which peripheral anterior synechia was absent.

The prognosis for most patients with essential iris atrophy is slow progression with eventual involvement of visual acuity caused by corneal edema, secondary glaucoma, or both. Sixty-three patients (77%) in the present study required treatment for increased IOP. In some cases, the treatment was to relieve corneal edema and in other cases it was to prevent glaucomatous atrophy of the optic nerve head.

Surgical intervention was required in 36 (44%) of our cases. This was indicated for the control of glaucomatous field loss in half of our cases and for the control of corneal edema in the remainder. Filtering surgery controlled the IOP in 69% of the reported cases, although the corneal edema was controlled in only 42%. These success rates are worse than for most glaucomas, but a standard filtering procedure is still the best choice when medical management is no longer adequate.

SUMMARY

We studied 82 cases of essential iris atrophy. The findings support some traditional concepts of this complex spectrum of disorders, but conflict with others.

Corneal abnormality appeared early and may be the primary disorder. A corneal endothelial disturbance was present in 55% of cases and corneal edema was present in 50%. Peripheral anterior synechiae occurred in all but one of the cases studied by gonioscopy. The pupil

was distorted in 71% of the cases and was usually pulled in the direction of the most prominent synechia. The iris was stretched with stromal atrophy in 71%. Iris holes occurred in 33%. The degree of corneal and iris changes occurred as a spectrum of disorders.

The prognosis for most patients with essential iris atrophy is slow progression with eventual involvement of vision because of corneal edema, secondary glaucoma, or both. Treatment of increased IOP was required in 77% of our cases, and 44% required surgical intervention.

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ENDOTHELIAL CELL LOSS DURING PENETRATING KERATOPLASTY

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In 1968, Maurice¹ first described the specular microscope for viewing the endothelium of the intact cornea in vitro. The instrument has been subsequently modified by Laing, Sandstrom, and Leibowitz² and by Bourne and Kaufmar³ in order to develop an instrument for use in the routine examination of the corneal endothelium. In 1976, Bourne⁴ described a further modification of the specular microscope, with a specialized corneal storage container, permitting a view of the endothelium of donor corneas before their transplantation. This technique of donor examination has enabled efficient screening of donor corneal tissue for subtle endothelial abnormalities. Additionally, one may compare the appearance of the donor endothelium after its transplantation, thus allowing an estimate of the endothelial damage that occurred at keratoplasty. Most damaged endothelial cells in humans are not replaced by cell division, but, rather, the endothelium heals by enlargement of the remaining cells⁵: thus, an estimate of the cell loss from keratoplasty can be made by comparing the average number of central endothelial cells per square millimeter in the donor cornea before and after its transplantation. In this study, we describe the endo-

thelial cell loss encountered in a series of 27 clear corneal transplants.

SUBJECTS AND METHODS

We studied the endothelium of 27 clear, penetrating corneal transplants before and after transplantation (Table). Examination of all donor corneas was performed before keratoplasty by use of a special corneal storage container4 in which the cornea was kept in McCarey-Kaufman⁶ medium at 4° C. All donor eves were removed within seven hours of the death of the donor, and all corneas were placed in McCarev-Kaufman medium containing 100 units per milliliter of penicillin-streptomycin solution within eight hours after enucleation. We observed the endothelium of these donor corneas with a modified specular microscope without opening the storage container. The shortened dipping cone on the modified objective lens4 remained fixed in a fully tightened position for all endothelial and calibration photographs. The donor corneas were stored in the McCarev-Kaufman medium for periods ranging from 6 to 63 hours (mean, 37 hours). Several corneas were refused as donors on the basis of their specular microscopic pattern alone.

Nineteen of the grafts were 7.5 mm. four were 8.0 mm, three were 8.5 mm, and one was 9.0 mm in diameter. Two additional penetrating grafts were performed during the same time period, but these did not remain clear. The donor corneal buttons were punched from the endothelial side by use of a corneal trephine press7 with the same trephine that was used on the recipient cornea. Subcon-

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From the Sections of Ophthalmology (Dr. Bourne) and Medical Research Statistics (Dr. O'Fallon), Mayo Clinic and Mayo Foundation, Rochester, Minnesota. This investigation was supported by the Mary and Alexander P. Hirsch Award of Fight for Sight, Inc., New York City. Portions of this study were presented to the American Medical Association Annual Clinical Convention, Section of Ophthalmology, San Francisco, California, June 19, 1977.

ENDOTHELIAL CELL LOSS IN PENETRATING KERATOPLASTY TABLE

	Recipi	s) Diagnosis	5 Traumatic corneal scar	•		로디) Fuchs' dystrophy 3 Interstitial keratitis		2 Fuchs dystrophy 5 Fuchs dystrophy 1 Interestial	•	ĀĀ	1 Keratoconus 9 Aphakic bullous keratonathy	CY.	N III			78 Fuchs' dystrophy	-	37 Keratoconus
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	Corneal Thickness	t(mm)	0.55	0.70	0.62	0.59	0.62	0.61	0.72		0.66	0.60	0.61	0.60	0.65 0.59	0.60	0.66	0.62	0.62
	Graft Size	(mm)	7.5	7.5	73	1, 1, 10, 10,	77.75	9.0	10 10 to	3	8.0	7.5	8.5	& 1. ໝີ່ໜີ່ກ	7.5	8.0 7.0	- ក្ វិសិទ	7.5	7.5
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nor helial	er mm	rostop- erative	3,675	2,493	2,913	2,290 1,910	2,487 2,131	2,239	3,113 3,195	3,156	2,289 2,027	3,137 1,993	2,207	1,812 2,046	2,138 1,631 2,034	1,758	1,431	1,212 990	385
Donor	Cells per mm	Preop- erative	3,486	2,439	2,976	2,381 1,998	2,628 2,233	2,374	3,374	3,552	2,578 2,487	3,872 2,634	2,986	2,472 2,878	3,070 2,456 3,141	2,686	2,190 2,477	2,536 2,449	2,888
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*Day of examination was first postoperative specular microscopic examination.

10 indicates comea removed from donor eye in situ and placed directly in McCarey-Kaufman medium.

1At first postoperative specular microscopic examination.

\$\{\creat{Combined keratoplasty}}\$ and cataract extraction.

junctival corticosteroids were often given at the end of the procedure, and topical corticosteroids were used after operation if there was significant or undue inflammation.

Twenty-two of the 27 transplants were examined with the specular microscope within one week after keratoplasty. We examined the other five transplants at later dates because of increased thickness of the graft in the early postoperative period or patient difficulties in cooperating (one patient was only 5 years old). The central corneal thickness was also measured in each transplant by the same investigator, either with a Haag-Streit pachymeter and its Mishima-Hedbys modification⁸ or with the specular microscope. With proper calibration, the position of the dipping cone lens on the specular microscope when the endothelium is in focus provides an objective measure of the thickness of the cornea as applanated plus the tear layer between the lens and the corneal epithelium.9 When the corneal thickness was measured in 30 different patients by one of us (W.M.B.), first with the Haag-Streit pachymeter and then with the specular microscope, the mean difference between paired measurements was 0.008 mm (microscope measurement was greater, standard error of the difference, 0.0028).

The central endothelial cell density was computed by counting the endothelial cells in three different photographic fields of known area from one cornea and averaging the results. A stencil containing 32 rectangles of increasing size was placed over the negative of the endothelial photograph to be counted, and the largest rectangular area in which cell borders could be discerned was chosen. Partial cells were counted on two sides of the rectangle. The area of each rectangle was measured with the photographic negative of a glass micrometer slide filmed through the appropriate specular microscope. The

calibration photograph for the clinical specular microscope was taken with the dipping cone positioned for a normal corneal thickness (0.54 mm). The areas of the fields counted varied from 0.017 mm² to 0.055 mm² (mean, 0.036 mm²). The endothelial cell loss in percent was computed for each transplant by comparing the endothelial cell counts before and after keratoplasty. Different variables were compared by either Spearman's rank correlation corrected for ties or the rank-sum test; P<.05 was considered to be statistically significant.

Cell counting involves two stages: first, identifying a rectangular stencil area within which to count the cells and second, the actual counting of the cells. Consequently, the reproducibility of each of these stages must be examined. This was accomplished by the following study.

Three preoperative and three postoperative cell counts were made on each of the 27 patients. From the resulting 162 counts, a random sample of ten preoperative and ten postoperative counts was selected for the reproducibility study. The proper identifications of roll and frame to be counted were coded on a separate envelope for each of the 20 counts to be redone. Within each sealed envelope was a slip of paper containing the same information as on the outside of the envelope and also indicating which area had been used in the original count. Reproducibility then involved, first, without the envelope being opened, the choosing of the area which seemed to be the reasonable one and the recording of this area on the outside of the envelope. The envelope was then opened and the area that had actually been used for the original count was identified and used in an attempt to reproduce the original count.

Of the 20 rectangular areas selected, seven (35%) were exactly the same ones

that were originally used, and three others differed only slightly from the original. The remaining ten areas involved the selection of a stencil area that differed from the one originally selected by more than 16%. Interestingly, eight of the ten "good" fits involved preoperative films.

There was good agreement between the original and the repeated counts in relation to the identity line (Fig. 1). By using deviation from the identity line as the measure of variation, the standard deviation of the repeated counts about the line was 5.8 cells. This deviation seemed independent of the actual cell count, and only one of the 20 points fell outside two standard deviations. In 50% of the counts, the relative deviation (absolute difference in counts as a percent of the original count) was less than 5% and it was less than 10% in 75% of the cases. Here again, reproducibility was better for the preoperative counts than for the postoperative

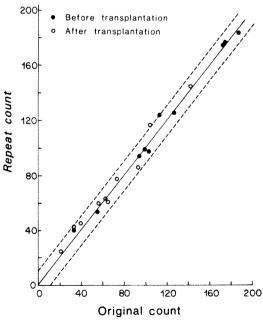


Fig. 1 (Bourne and O'Fallon). Cell count reproducibility. The dashed lines indicate two standard deviations of the repeat counts from the identity line. The reproducibility is good.

counts. For example, in the former instance, 50% of the second counts differed from the original by less than 1.5%, whereas in the latter, all replicates differed from the original by more than 1.5%.

These data illustrate an acceptable level of reproducibility for the actual cell counts. The more subjective selection of a stencil area to guide the counting was less well-replicated, but this seems less critical. The data indicate a better level of reproducibility among the preoperative counts than among the postoperative counts where the trauma and cell loss make it difficult to obtain a clear image of the cell structure for counting.

RESULTS

A typical donor endothelium before and after its transplantation (Case 6) is shown (Fig. 2). The results of the endothelial studies in all 27 patients are shown (Table). Many different variables were studied for possible correlation with cell loss. There was a significant association between the presence of a lens postoperatively and the cell loss occurring at keratoplasty (P<.01). Thus, aphakic transplants (all eyes that were aphakic after transplantation) had significantly less endothelial cell loss from the keratoplasty

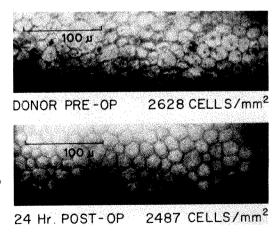


Fig. 2 (Bourne and O'Fallon). The donor endothelium in Case 6 before and after transplantation.

procedure than did phakic grafts (Fig. 3). This was true whether or not the five patients examined with the clinical specular microscope for the first time more than one week after keratoplasty were included in the analysis. The cell loss in eyes that were aphakic before the procedure did not differ significantly from that in eyes undergoing combined penetrating keratoplasty and intracapsular cataract extraction. The phakic and aphakic grafts also differed in three other (probably unrelated) aspects: recipient age (aphakic older, P<.01), corneal thickness at first postoperative week (aphakic thicker. P<.05), and graft size (aphakic smaller, P<.05). Because of the large difference in phakic and aphakic grafts, the two groups were analyzed separately. In phakic transplants, less cell loss was associated with larger grafts (P<.01) and shorter death to enucleation times (P=.05). When the phakic and aphakic groups were separated, no significant association was demonstrated between endothelial cell loss and preoperative cell count, donor age, storage times, vitrectomy, or corneal thickness during the first postoperative week.

The preoperative cell count was related to donor age (younger donors had more

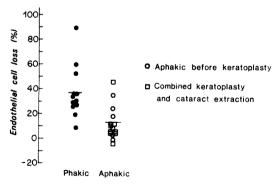


Fig. 3 (Bourne and O'Fallon). Endothelial cell loss during penetrating keratoplasty. Cell loss was greater during phakic procedure (mean loss, 37%) than during the aphakic combined procedures (mean loss, 12%).

cells, P<.01). A similar correlation between age and endothelial cell density has been reported previously.^{3,10} Multiple regression analysis showed that the single most important variable affecting cell loss was the presence of a lens postoperatively (phakic vs aphakic). A second significant, but less important factor was the graft size.

DISCUSSION

The mean endothelial cell loss that occurred during the 27 transplants was 23%. This was surprisingly low and less than the endothelial cell loss occurring during intraocular lens implantation reported recently.11 The mean endothelial cell density for the 27 corneal transplants postoperatively was 2,152 cells per square millimeter. This is also nearly double that of several previously reported series of endothelial cell counts in corneal transplants.12-14 The low endothelial cell loss shown in this study may be the result of improved surgical techniques or better methods of donor tissue preservation, or both. The fairly young donor tissue used (mean age, 34 years) also may have been a factor. Additionally, most of the grafts studied previously were studied more than one year after keratoplasty, and many more transplanted endothelial cells could be lost with time. This may be true. especially with aphabic grafts, which had less endothelial cells than phakic grafts in a previous longterm study, although the difference was not significant when corrected for recipient age.14 Further cell loss has not occurred to date in most of the grafts in this study (up to one year after operation).

The finding of less endothelial cell loss in aphakic corneal transplants is reasonable from a mechanical point of view and may be caused by the deeper anterior chamber present during the operation; this causes less damage to the donor endothelial cells from rubbing against the recipient iris and lens. A similar mechanism may explain the lower cell loss in grafts of larger size. That the phakic transplant with the least cell loss (Case 8) had an enlarged 15-mm cornea from congenital glaucoma supports this explanation. A 9-mm transplant was performed, and the postoperative depth of the anterior chamber was 4.0 mm, much deeper than the normal phakic anterior chamber depth of 3.2 mm.¹⁵ Additionally, the two aphakic transplants with the most endothelial cell loss each had complicated procedures. One (Case 21) had a trabeculectomy and a large intraocular epithelial cyst removed during the procedure; the other (Case 24) had an intraoperative massive choroidal hemorrhage that was controlled by multiple posterior sclerotomies and rapid fixation of the donor graft to prevent the intraocular contents from being expelled from the eye. This finding of less endothelial cell loss in aphakic corneal transplants confirms the results of a previous report⁵ comparing two patients with mated donor corneas. Surgical techniques designed to deepen the anterior chamber during keratoplasty^{16,17} may decrease the endothelial cell loss in these procedures; however, we have not found these techniques useful.

More cell loss occurred in donor corneas that had longer intervals from death to enucleation. More endothelial cell damage occurs with longer exposure to postmortem aqueous humor, 18,19 and such corneas may be more susceptible to cell loss.

Aphakic corneal grafts were thicker than the phakic transplants during the first postoperative week. This confirms our clinical impression, and the thickening occurred despite the greater endothelial cell loss in the phakic transplants. The cause of the increased corneal thickness in aphakic keratoplasties is not known. The postoperative intraocular pressure (IOP) is usually higher in apha-

kic transplants,²⁰ and this alone tends to make them thinner. Vitreous humor decreases endothelial fluid transport,²¹ and access to the vitreous may account for the increased corneal thickness in aphabic transplants.

We were surprised not to find a significant correlation between endothelial loss and graft thickness in the first postoperative week when the phakic and aphakic groups were studied separately. Any variation in corneal thickness reflecting decreased endothelial function may have been masked by a different variation in postoperative IOP, which was not analyzed.

SUMMARY

We studied the central donor endothelium of 27 clear, penetrating corneal transplants with the specular microscope before and after keratoplasty. We examined the donor corneas first, in vitro, while they were immersed in McCarev-Kaufman preservation medium before transplantation. This preoperative examination proved valuable for screening donor corneas, several of which were not used on the basis of the specular microscopic appearance alone. We examined the donor endothelium again within one week after keratoplasty and calculated the number of endothelial cells per square millimeter from photographs. The reproducibility of the counting method was acceptable. Comparison of the examinations before and after transplantation on each patient showed that, on the average, 23% of the donor endothelial cells were lost during keratoplasty. The 12 phakic transplants lost significantly more endothelial cells than did the 15 aphakic grafts (37% vs 12%). A possible explanation for the increased cell loss in phakic keratoplasties is the shallow anterior chamber present during the initial placement of the graft. Phakic grafts that were larger or had shorter time intervals between donor death and enucleation lost fewer cells. Aphakic transplants had larger postoperative corneal thicknesses than did phakic transplants.

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PULSATING METASTATIC TUMOR OF THE ORBIT

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Most pulsating orbital tumors are caused by vascular malformations, arteriovenous fistulas, or cerebrospinal fluid pulsations transmitted through major developmental or postsurgical defects in the orbital bones. But the signs in the patient reported herein indicate that a metastatic renal deposit in the orbit can be so well vascularized as to generate its own pulsations.

CASE REPORT

A 47-year-old white man in apparent good health noted the onset of swelling of the right upper eyelid and diplopia that had been increasing gradually for six weeks (Fig. 1). Ocular examination showed uncorrected visual acuity of 6/6(20/20) in each eye. A large pulsatile, collapsible mass involved the right temple, as well as the upper outer quadrant of the right orbit. The right globe was proptosed 5 mm and displaced downward and inward. The conjunctiva was chemotic, and partial blepharoptosis of the right upper eyelid was present. The pupils were reactive, but ductions of the right eye were limited in elevation and abduction. The intraocular pressures and fundi were unremarkable.

Results of general physical examination, routine chest x-ray studies, electrocardiogram, urinalysis, and hematologic studies were all within normal limits. Routine x-ray films and tomograms of the skull, orbits, and paranasal sinuses showed extensive bone destruction involving the superolateral border of the right orbit in the region of the lacrimal fossa (Fig. 2). The destruction extended posteriorly to involve the linea innominata. The bony erosion was sharply demarcated, had the appearance of a slowly growing mass, and thus suggested a lacrimal gland tumor. B-scan ultrasonography revealed a mass consistent with a vascular tumor that transmitted sound well with few internal echoes. It measured about 10 mm anteroposteriorly and 20 mm horizontally; it followed the course of the superolateral orbital wall from the region of the posterior

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Fig. 1 (Howard and associates). Clinical photograph showing proptosis of the right globe, with displacement downward and inward. A collapsible pulsatile mass involved the right temple and the right upper eyelid.

pole of the globe to the orbital apex. Computed tomography disclosed involvement of the superolateral portion of the orbit with extension into the temporal fossa through a defect in the lateral orbital wall (Fig. 3).

At the time of orbitotomy, a large encapsulated tumor was easily located along the lateral wall of the orbit; it extended back to the apex of the orbit and had invaded the overlying anterolateral orbital bone. There was a prominent vascular network on the surface which penetrated into the substance of the mass. Bleeding during surgery was profuse, and a large hemorrhagic cavity in the neoplasm was discovered. It was not possible to remove the mass entirely, so we elected to amputate the visible portion. Pathologic evaluation revealed the tumor was a metastatic renal cell carcinoma.

We continued laboratory investigations. Radiologic study of the long bones and radioactive scanning showed no evidence of metastases; radionucleotide scanning of the liver and spleen was

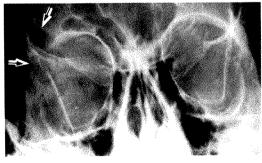


Fig. 2 (Howard and associates). Anteroposterior skull film showing destruction of the superolateral border of the right orbit in the region of the lacrimal fossa (arrows).

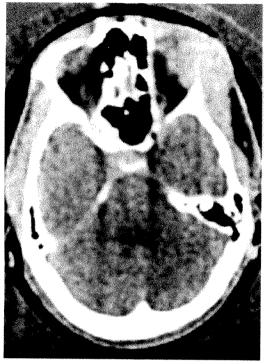


Fig. 3 (Howard and associates). Computerized tomographic study through the orbits reveals a destructive lesion of the anterolateral portion of the right orbital wall; the lesion extends into the temporal fossa

likewise negative. The intravenous pyelogram revealed a mass involving the middle pole of the left kidney, without obstruction of the ureter. Left renal arteriograms confirmed the presence of a mass measuring 12 cm in the craniocaudal dimension, and 10 cm in width. Right carotid arteriography demonstrated a markedly vascular tumor occupying the anterior two thirds of the right orbit, and extending toward the roof and right frontal sinus. The vascular supply to the orbital tumor transported through a moderately enlarged ophthalmic branch of the internal carotid artery, and from ethmoid branches of the internal maxillary branch of the external carotid artery (Fig. 4).

Two weeks after orbitotomy, we treated the patient with a course of radiotherapy to the right orbit consisting of 3,500 rads administered in divided doses during a three-week period. He was then admitted to the Sloan-Kettering Memorial Cancer Center in New York City, where a left nephrectomy was performed. Postoperatively, immunotherapy was begun by using extracts of his renal tumor that were prepared for reinjection to stimulate an auto-immune response to the carcinoma. Nine months after orbitotomy, he has shown a satisfactory local response to the radiotherapy for his orbital metastasis, and orbital pulsations have totally disappeared. The patient has resumed full activity, feels subjectively well, and no further sites of metastatic disease

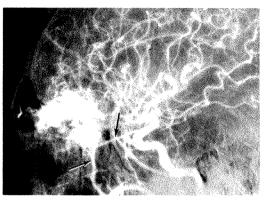


Fig. 4 (Howard and associates). Arteriogram shows well vascularized orbital mass fed by ophthalmic artery (upper arrow) and a branch of the internal maxillary (lower arrow).

have been discovered. The outlook, of course, is guarded.

RESULTS

On microscopic examination, the tumor was circumscribed, but not encapsulated. and it infiltrated the surrounding orbital tissues in small nests. The main mass of the tumor was composed of a single population of cells. They were subdivided into variably sized lobules by fibrovascular septa (Fig. 5). On routine hematoxylin and eosin sections, the tumor cells appeared large and clear (Fig. 5, inset); they formed solid nests and possessed irregularly shaped hyperchromatic nuclei with prominent nucleoli. Mitotic figures were moderately common. No manifest lumen formation was demonstrable, although central areas within the nests showed necrosis with resultant pseudoglandular formations. The tumor cell cytoplasm stained positively with the PAS technique, and this PAS-positive material was diastase labile. Tumor cell emboli were found in some of the capillary lumina within the fibrous septa.

Electron microscopic studies were performed on fresh tissue obtained at the time of surgery and placed in glutaraldehyde. Three types of cells were identified. The predominant cell was polyhedral, displaying electron-lucent cytoplasm

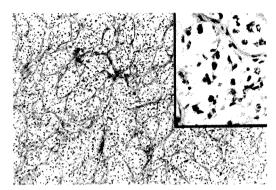


Fig. 5 (Howard and associates). Tumor is composed of clear cells and subdivided into lobules by fibrovascular strands. Inset, clear cytoplasm and connective tissue septa. (hematoxylin and eosin, × 63: inset, × 475).

with scattered mitochondria and only a few short profiles of rough surfaced endoplasmic reticulum (Fig. 6, top). The cytoplasm of these cells was dominated by myriad dark-staining glycogen granules (Fig. 6, top inset). The second type of cell had a more abundant rough-surfaced endoplasmic reticulum and more numerous mitochrondria, but less glycogen. The third type of cell was a variant of the second, additionally containing vacuoles (Fig. 6, bottom). While no frozen sections were performed, because of lack of wet tissue, the clear appearance of these vacuoles is most consistent with dissolved-out lipid. No lumen formation was discovered, and only rare intercellular junctions consisting of poorly developed desmosomes were found. Basement membrane formation surrounded the tumor cells where they abutted on the fibrovascular stroma. Occasional intracytoplasmic lumina with villous formations were found (Fig. 6, bottom inset), but lateral intercellular interdigitations or complex infoldings of the basilar regions of the cells were not seen.

Discussion

If maldevelopmental anomalies involving the orbital bones (such as occur in neurofibromatosis and meningoencephaloceles) and major postoperative defects in the orbital bones are excluded,¹ the only recognized causes of pulsating exophthalmos have been vascular tumors and fistulas? In our experience, the present metastatic tumor is unique. Patients with acquired pulsatile exophthalmos accompanied by evidence of orbital bone destruction should be suspected of having a well-vascularized metastatic tumor, such as renal cell carcinoma.

Pulsatile exophthalmos of arterial origin should be distinguished from positional and intermittent exophthalmos.2 In positional exophthalmos, proptosis can be brought out by having the patient bend forward or perform a Valsalva maneuver; this condition almost always signifies a venous malformation or varix that forces blood to collect in collapsible abnormal spaces. Intermittent exophthalmos is neither voluntarily produced nor constantly present because it is not dependent on arterialization or positional changes. Rather, it reflects variations in the degree of exophthalmos caused by a fluctuating inflammatory component to the lesion, such as hemorrhage into lymphangiomas, ruptured dermoids, and expanding and contracting mucoceles.

The palpable pulsations in the present patient have been interpreted by us as the consequence of the tumor's parasitization of two arterial supplies, namely, the ophthalmic artery and terminal branches of the internal maxillary artery of the external carotid system. At the time of surgery, leashes of wormy vessels were discovered surrounding the pseudocapsule of the tumor and diving into its substance. That the orbital bone destruction allowed transmission of cerebrospinal fluid pulsations seems a much less likely explanation for the pulsations because most of the orbital roof and the posterior portion of the sphenoid were intact. The osseous destruction was centered predominantly in the area of the lacrimal gland, the supralateral orbital rim, and the lateral orbital wall. The most compelling reason

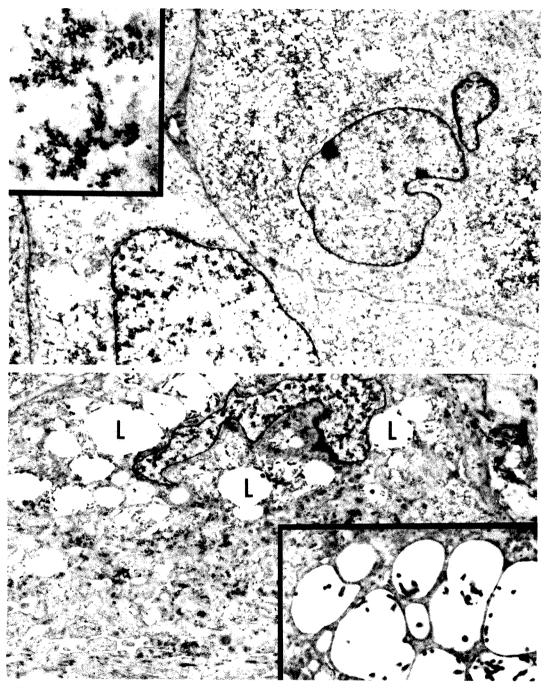


Fig. 6 (Howard and associates). Top, Polygonal tumor cells have electron-lucent cytoplasm containing myriad darkly staining glycogen granules shown also in inset. Bottom, Tumor cells with vacuoles consistent with dissolved-out lipid (L). Inset, pseudolumenal cytoplasmic structures with villi (top, \times 4,600; inset, \times 25,700; bottom, \times 5,200; inset, \times 8,300).

for attributing the tumor's pulsations to its intrinsic vascularity is that no pulsations have been noted since surgical excision and radiotherapy, whereas transmitted cerebrospinal fluid pulsations should not have been affected by these treatments.

Ferry and Font³ studied a series of 227 patients with carcinomas metastatic to the eve and orbit; of these, nine patients had metastases that originated in the kidney. In eight of these nine patients, the primary tumor was undetected before the ophthalmic metastasis. The average interval to subsequent discovery of the primary renal carcinoma was seven months long. Two of 28 patients with metastases exclusively to the orbit had unknown primary tumors in the kidney.3,4 Cutaneous metastases may likewise herald an unrecognized renal cell carcinoma.⁵ In our patient, the histopathologic interpretation of the lesion led to systemic laboratory tests and the immediate discovery of a left renal primary tumor. Among metastatic tumors, renal cell carcinoma is highly characteristic under the microscope; it is composed of clear cells regularly compartmentalized by fibrovascular trabecula. The electron microscopic studies disclosed the diagnostic combination of large amounts of glycogen granules and cytoplasmic lipid droplets in some of the tumor cells.⁶ A low degree of differentiation was exemplified by the rarity of desmosome formation, the general lack of surface specializations, lateral interdigitations, basal infoldings, and the lack of polarity and lumen formation.

Pulsating cutaneous renal cell metastases have been described,⁷ but to our knowledge this is the first report of a pulsatile metastatic orbital tumor. Preoperatively, the diagnosis of metastatic orbital renal cell carcinoma should be suspected in patients who have an acquired pulsatile tumor with extensive bone destruction demonstrated on computed tomography and routine radiographic studies. Virtually no primary orbital tumor

produces, early in its course, the enormous bone destruction so often seen with metastatic tumors.

SUMMARY

A 47-year-old white man in apparent good health had diplopia and swelling of the right upper eyelid. Ocular examination showed proptosis of the right eye, together with a large, pulsatile, collapsible mass simulating a vascular neoplasm and involving the right temple as well as the right upper outer quadrant of the orbit. Biopsy of the orbital tumor disclosed a clear-cell carcinoma compatible with a renal primary tumor; subsequent laboratory examination revealed the offending tumor in the left kidney. Renal carcinomas may metastasize to the globe or to the orbit before the primary tumor is recognized. Pulsatile exophthalmos acquired in middle life associated with significant bone destruction represents a constellation of findings most consistent with a metastatic tumor, probably renal carcinoma, caused by the exceedingly rich vascularization of these metastatic deposits.

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SURGICAL MANAGEMENT OF EPITHELIAL INGROWTH

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Epithelial invasion of the anterior chamber is a rare, potentially disastrous complication following anterior segment surgery and penetrating ocular injuries.^{1–4} Epithelial invasion can occur in three forms⁵: (1) "pearl" tumors of the iris, (2) an epithelial cyst in the anterior chamber, and (3) growth of epithelium as a sheet on anterior chamber structures and posteriorly over the ciliary body and retina. The latter form is referred to as epithelial downgrowth or epithelial ingrowth (Figs. 1 and 2). This form has the worst prognosis and is the subject of this report.

Various modes of therapy have been attempted for epithelial invasion of the anterior chamber including radiation 6-11

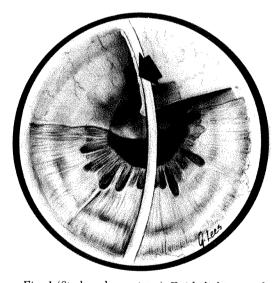


Fig. 1 (Stark and associates). Epithelial ingrowth involving posterior corneal surface (arrow), superior iris, and anterior vitreous face.

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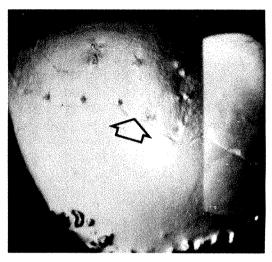


Fig. 2 (Stark and associates). Retroillumination appearance of epithelial sheet on the posterior corneal surface. Advancing epithelial edge is thickened and irregular (arrow).

and surgical methods. 12-16 Radiation therapy has been abandoned, and surgical treatment has been successful in up to 27% of reported cases. Maumenee¹⁵ reported the only large series with surgical treatment; in 11 of his 41 cases (27%) final visual acuity of 6/15 (20/50) or better was achieved with controllable intraocular pressure and without evidence of recurrence of the epithelial invasion. Treatment included photocoagulation of the anterior iris surface, excision of the involved iris, chemical treatment or cryotherapy to the involved portion of the posterior corneal surface and other involved tissues, and identification and closure of the fistula.

This paper presents our current surgical technique and results in ten consecutive cases of epithelial invasion of the anterior chamber operated on during the past 30 months.

MATERIAL AND METHODS

Subjects—We examined ten consecutive patients with uniocular epithelial ingrowth involving the anterior chamber between May 12, 1975, and May 20, 1976 (Table). This group included five men and five women, ranging in age from 33 to 85 years. Nine patients had undergone previous cataract surgery on the affected eye, and a traumatic iris cyst had been removed in the remaining case. Five patients had normal or abnormally low intraocular pressure and a fistula was demonstrated by the Seidel test. No fistula was demonstrated in the other five patients and the intraocular pressure was increased.

The extent of epithelial involvement of intraocular tissues varied considerably. In each affected eye the cornea, anterior chamber, and iris were involved. The extent of iris involvement ranged from 30 to 100%. Epithelium covered the vitreous face in eight eyes and extended posteriorly onto the ciliary body in three eyes. Preoperative visual acuity ranged from 6/12 (20/40) to light perception.

Surgical technique—Preoperatively, the iris was treated by argon laser photocoagulation to define the extent of epithelial involvement.17 Photocoagulation spots of 500 μ were placed along the advancing edge of the epithelial sheet in such a way that one half of each spot turned white, indicating the presence of epithelium, and the other half turned brown, indicating noninvolved iris tissue (Fig. 3). In several cases, the entire area of involved iris was photocoagulated. Photocoagulation was performed within 24 hours of the planned surgical procedure because this treatment can cause moderate anteriorchamber inflammation.

Traction sutures were placed under each rectus muscle in a transconjunctival fashion or after a 360-degree conjunctival peritomy was performed. A limbal-based conjunctival flap was dissected to expose the superior corneoscleral limbus. Two percent fluorescein dye was applied, together with external pressure to identify aqueous leakage at a fistula (Fig. 4). If a fistula was identified, it was closed with sutures; or a one-half thickness scleral flap, hinged anteriorly, was prepared to cover the fistula later in the operation¹⁸ (Fig. 5). Bowman's membrane was scarified with a knife to mark the advancing edge of the epithelial sheet on the posterior surface of the cornea.

When a pars plana approach was used. a sclerotomy was performed 4 mm posterior and parallel to the corneoscleral limbus in the superotemporal quadrant. The vitrectomy instrument, equipped with an overlying fiberoptic sleeve, was inserted into the pupillary space. The area of involved vitreous and iris was excised, a bite at a time, by using an oscillating cutting mode to minimize traction on the iris root (Fig. 6). If bleeding occurred, it was controlled by temporarily increasing the intraocular pressure or by applying bipolar diathermy. 19 We tried to aspirate all excised material into the cutting port, and this tissue was studied cytologically to confirm the clinical diagnosis of epithelial ingrowth.

After excising the involved vitreous and iris tissue, any vitreous gel remaining in the anterior one half of the vitreous cavity was removed. This provided a fluid space that could subsequently be filled with air to enhance the freezing technique. The vitrectomy instrument was then withdrawn and the sclerotomy site closed with multiple sutures.

The anterior one half of the eye was then filled with sterile air by exchanging the intraocular fluid by means of a needle attached by a three-way stopcock to an aspiration syringe and a syringe filled with air. Alternatively, fluid-gas exchange was perforned before withdrawing the vitrectomy instrument. Air was intro-

TABLE
EPITHELIAL INGROWTH CASES*

	Follow-up, mos	33	26	21	21	20	22	55	20	18	53
	Additional Surgery	None	Repair of Retinal Detachment	None	PK	PK	None	None	PK	None	PK
	Macular Edema	Yes	°Z	No	No edema	Yes	No	Yes	Yes	No	Yes; Macular pucker
	Postoperative Macular Additional Follow-up, Complication Edema Surgery mos	None	Retinal Detachment 4 mos.	None	Corneal	Corneal edema	None	Hypotony	Corneal edema	None	Corneal edema
	Acuity	6/24 (20/80)	6/12 (20/40)	6/12 (20/40)	5/2004	6/60 (20/200)	6/12 (20/40)	LP	6/60 (20/200)	6/9 (20/30)	6/12 (20/40) 6/60 (20/200)
	Visual Acuity Postoperative	6/60 (20/200) 6/24 (20/80)	6/30 (20/100)	6/90 (20/300) 6/12 (20/40)	3/200	LP	6/18 (20/60)	6/18(20/60)	HW	6/15 (20/50)	6/12 (20/40)
	Fistula	Yes	S O	No	Yes	No	No	Yes	No	Yes	ON
tent (%)		No	Š	No	Yes		Yes	Yes	No	No	°Z
and Ex	Vitreous Ciliary Face Body	Yes	Yes	Yes	Yes	Phakic	Yes	Yes	Yes	No	Yes
nvolved	Iris	20	33	33	80	100	100	33	33	33	40
Fissues Involved and Extent (%)	Anterior Chamber Angle	20	33	33	08	100	100	33	33	33	40
-	Cornea	33	33	33	33	100	33	33	33	33	33
Previous Surgery	(Mos Before Diag- nosis of Epithelial Ingrowth)	ICCE, 2	ICCE, 4	ICCE, 36	ICCE, 10	Traumatic iris cyst, 75	ICCE, 10	ICCE, 5	ICCE, 2	ICCE, 2	ICCE, 15
	Case Age No., (yrs) Sex	т. Н	M M	<u>ir</u> ,	ír.	M.	M	X	M.	<u> </u>	E.
***************************************	Case Age No., (yrs)	77,	50,	64	85,	33,	55,	68,	59,	70,	51,
	Cas No.	j.,	વ્ય	ω,	Ą	rζ	6,	7,	∞ ʻ	တ်	10,

*ICCE designates intracapsular cataract extraction; PK, penetrating keratoplasty. †Extensive glaucomatous cupping of the optic disk.



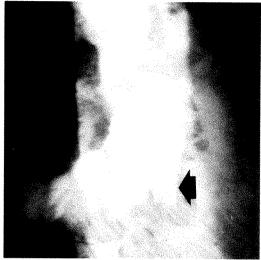


Fig. 3 (Stark and associates). Case 1. Left, Argon laser treatment to delineate the extent of epithelial growth on the anterior iris surface. Photocoagulation caused the epithelium to turn white and noninvolved iris tissue turned brown. Right, Higher magnification of junction between the epithelium (arrow) and adjacent noninvolved iris.

duced through the infusion system of the instrument while intraocular fluid was passively displaced through the aspiration system vented to the atmosphere.

If a large fistula was present, the partial-thickness scleral flap was reflected over the fistula and sutured to the peripheral cornea. The fundus was examined by indirect ophthalmoscopy with scleral depression. Any retinal tears were treated with cryotherapy and their position marked on the sclera. A silicone-rubber exoplant provided a scleral buckling effect beneath any retinal break.

Cryotherapy was then applied in a transcorneal and transceleral fashion to devitalize the epithelium remaining on the posterior surface of the cornea, in the anterior chamber angle, and on the ciliary body (Fig. 7). The intraocular bubble

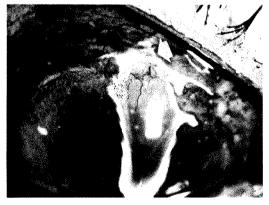


Fig. 4 (Stark and associates). Case 4. Aqueous humor leaking through a fistula (arrow) flows across the cornea as demonstrated by fluorescein dye and blue light.

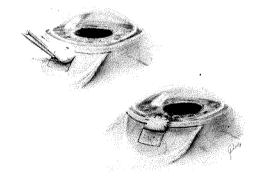


Fig. 5 (Stark and associates). A partial-thickness scleral flap, hinged anteriorly, is used to cover the external opening of the fistula if it cannot be closed with sutures. (From Rice, T.R., and Michels, R.G.¹⁸)

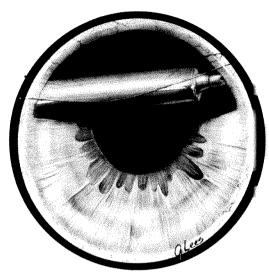
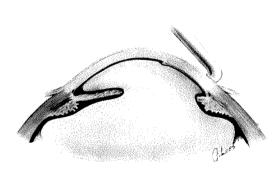


Fig. 6 (Stark and associates). The area of iris and vitreous involved by the epithelial ingrowth is excised with the vitrectomy instrument except for the peripheral iris.

provided a thermal insulating effect that caused each freeze to persist longer and also provided improved control over the size of each cryo-application. One can advance the full-thickness freeze from the corneoscleral limbus onto the cornea with considerable precision and extend the freeze just beyond the edge of the epithelial sheet, thus minimizing damage to the adjacent corneal endothelial cells. A freeze-refreeze technique was used to assure destruction of the epithelial cells. Part of the air bubble was then removed and replaced by physiologic solution if no retinal breaks were present.

Postoperatively, the eye was treated frequently with topical corticosteroids. Posturing of the patient was used, if retinal breaks were present, to position the remaining intraocular bubble against the retinal breaks.





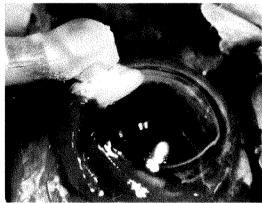


Fig. 7 (Stark and associates). Top left, Air bubble fills the anterior one half of the eye and provides a thermal insulating effect to enhance cryotherapy applied in a transcorneal and transscleral fashion. Top right, The full-thickness freeze can be advanced onto the cornea with considerable precision in order to treat the area of epithelial involvement and minimize damage to adjacent non-involved tissue. Bottom left, Areas of ciliary body involvement by epithelial ingrowth are treated with transscleral cryotherapy. Ice is visible on the ciliary processes (arrow).

RESULTS

Microscopic examination of excised intraocular tissue confirmed the diagnosis of epithelial ingrowth in each case. No intraoperative or early postoperative complications occurred. Except in Case 5, which required freezing of the entire cornea, all corneas were clear immediately after surgery. The epithelial sheet on the back of each cornea appeared white on the first postoperative day and began to slough on the second postoperative day (Fig. 8). The epithelial sheet usually disappeared by the fifth postoperative day, leaving only faint stromal haze.

Follow-up after surgery ranged from 18 to 33 months, with an average follow-up of 23 months. Visual acuity improved after surgery in eight of ten cases, and four eyes achieved final vision of 6/12 (20/40) or better. Intraocular pressure remained less than 21 mm Hg in all cases although two eyes required topical antiglaucoma medications. In one eye (Case 7) the intraocular pressure was 6 mm Hg.

Decompensation of the central cornea occurred in four eyes between two weeks and eight months after surgery and required penetrating keratoplasty. Histopathologic examination of these corneal buttons demonstrated residual intraocu-

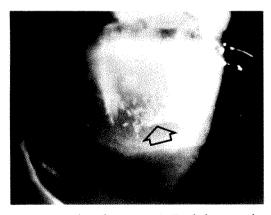


Fig. 8 (Stark and associates). Epithelium on the posterior surface of the cornea (arrow) turns white one day after cryotherapy and begins to slough.

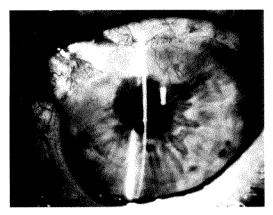
lar epithelial cells in one case (Case 5). This eye had epithelial involvement of the entire anterior chamber and probably the posterior chamber, before the operation for epithelial ingrowth. The corneal graft in this eye has remained clear for one year, and vision is correctable to 6/60 (20/200). The three other corneal buttons had a retrocorneal fibrous membrane without evidence of residual intraocular epithelium. All four corneal grafts remained clear throughout the follow-up interval.

The intraocular media were clear at the time of the last examination in nine of ten eyes, including the four eyes requiring penetrating keratoplasty. Except for Case 5, none has shown histologic or clinical evidence of residual viable intraocular epithelium (Figs. 9-11). Cystoid macular edema accounted for the reduced acuity in three of six eyes with postoperative vision of less than 6/12 (20/40) (Cases 1, 5, and 8). In one other (Case 7), vision was less than 6/12 (20/40) because of hypotony. One eye had a vitreoretinal membrane affecting the macula (Case 10), and one eye had optic nerve damage from previous glaucoma (Case 4).

DISCUSSION

Previous reports have indicated that results of treating epithelial ingrowth were not encouraging, especially when intraocular involvement was extensive. 1-16 Maumenee 15 reviewed 40 histologically proven cases, and in 11 (27.5%) vision of 6/15 (20/50) or better was achieved after surgery. Postoperative complications were significant, including 20 eyes with diffuse corneal edema, five eyes with hypotony, and five eyes that required later enucleation.

Our modifications of Maumenee's technique offer several advantages, especially in severely affected eyes. In our series, at least one third of the posterior corneal



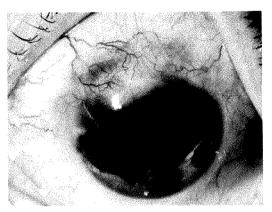


Fig. 9 (Stark and associates). Case 3. Left, Preoperative appearance showing epithelium involving the superior one third of the cornea (arrow). Right, Appearance 24 months after surgery. The central cornea is clear and the peripheral cornea is opaque and vascularized. Vision is limited by cystoid macular edema.

surface was involved with epithelium in every case, ranging from 33% to total involvement. Epithelium on the iris was present in every case and varied from 30% to total involvement. The vitreous face was involved by epithelium in eight eyes, and in three eyes epithelium clearly extended onto the ciliary processes. Despite the extent of epithelial involvement, 40% of our cases achieved postoperative vision of 6/12 (20/40) or better.

Instruments and surgical techniques designed for vitreous surgery provide a

"closed-eye" system and permit precise bite-by-bite control of tissue excision. Intraocular bleeding can usually be controlled by temporarily increasing the intraocular pressure or by use of bipolar diathermy. After removing the anterior vitreous gel, the intraocular fluid can be replaced by an air bubble that lines the anterior chamber, iris root, and ciliary body. The air functions as a thermal insulator and facilitates full-thickness freezes, which can be controlled to treat only the area of epithelial involvement. The intraocular air bubble also functions as a

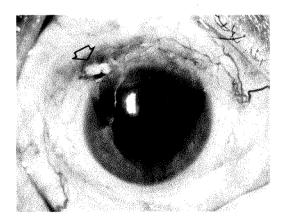


Fig. 10 (Stark and associates). Case 9. Postoperative appearance 12 months after surgery for epithelial ingrowth. A scleral flap (arrow) was used to cover the fistula.

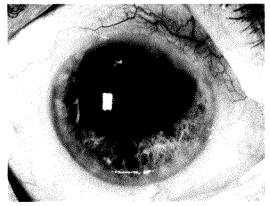


Fig. 11 (Stark and associates). Case 2. Postoperative appearance six months after surgery for epithelial ingrowth.

goniolens and permits freezing of the anterior chamber angle, iris root, and, if necessary, the adjacent ciliary body under direct visualization.

The insulating effect of air probably provides a more intense tissue freeze, although we have not measured the temperature by using a thermocouple. However, because in one of our cases hypotony developed after surgery, we have recently modified our cryotherapy technique. We now freeze the area of epithelial involvement only once, and do not use a freeze-thaw-refreeze cycle. Recently, we treated one patient with epithelial ingrowth by using this modification. The follow-up of this additional case is now four months, and the eye is not included in this series. However, in that case, the intraocular epithelium appeared white on the first postoperative day and sloughed from the cornea on the second and third postoperative days. This eye now shows no residual intraocular epithelium, and we tentatively recommend the singlefreeze technique, together with use of intraocular air.

Surgical management of epithelial ingrowth is easier, and the results should be better, if this condition is diagnosed and treated early. The advancing border of an epithelial sheet on the back of the cornea is typically several layers thick and has an irregular beaded margin. Usually the involved area of cornea remains clear. If an epithelial ingrowth is suspected, a Seidel test with 2% fluorescein and external pressure should be done to search for a fistula, which is present in about 20% of cases. Gonioscopy should be performed to identify possible iris involvement, and argon laser photocoagulation is useful to confirm the presence of epithelium growing on the anterior iris surface.

When the diagnosis of epithelial ingrowth is suspected, many physicians follow the area of corneal involvement before recommending surgery. In our ex-

perience the corneal involvement progresses more slowly than involvement of the anterior iris surface. Therefore, we encourage early diagnosis and prompt surgical treatment. If the diagnosis can be made when only a small area of the cornea and anterior chamber angle is involved, less extensive surgery may be possible. In five such cases seen during the past seven years, we have been successful in destroying the intraocular epithelium by transcorneal cryotherapy without intraocular surgery.

One potential complication related to the use of the vitrectomy instrument method is dispersion of epithelial cells throughout the eye. Cytologic examination of specimens removed in this series showed many free epithelial cells. We make every effort to remove all material aspirated into the cutting port, and we specifically avoid back-pressure through the aspiration system. We have only seen evidence of persistent intraocular epithelium in one case.

Although our follow-up averages 23 months (range, 18 to 33 months), we consider this to be a preliminary report. Additional cases with longer follow-up are needed to examine fully the value of this or other surgical methods. We believe, however, that this microsurgical technique, including use of intraocular air to facilitate external cryotherapy, offers significant advantages in the management of epithelial ingrowth.

SUMMARY

In ten consecutive cases of epithelial ingrowth operated on during the past 33 months, therapy included photocoagulation of the iris followed by excision of involved iris tissue and vitreous gel by means of instruments designed for vitreous surgery. Epithelium remaining on the posterior surface of the cornea, ciliary body, and in the anterior chamber was destroyed by controlled transcorneal and

transscleral cryotherapy. An intraocular air bubble was used to provide an insulating effect and a more effective, controllable freeze. All patients except two had improved vision postoperatively, and four of the ten patients had postoperative visual acuity of 6/12 (20/40) or better.

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CLINICAL MANIFESTATIONS OF BRAWNY SCLERITIS

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Brawny scleritis is an uncommon disease that can be difficult to diagnose. Retinal or choroidal detachments, or both, choroidal elevations, and intraocular inflammation can all occur with the scleral changes. ¹⁻³ We present herein four cases of brawny scleritis. In two of the cases, the eyes were removed after a diagnosis of choroidal melanoma. In the other two cases, recognition of characteristic clinical signs led to the correct diagnosis, despite ultrasonography and radioactive phosphorus (³²P) uptake tests, or both, which yielded findings indistinguishable from those in melanoma.

CASE REPORTS

Case 1—A 33-year-old white woman in good health was seen in the emergency room of the Massachusetts Eye and Ear Infirmary on Sept. 4, 1961. She had a one-week history of hazy vision in the lower field of her right eye, succeeded by a "curtain" in the superotemporal quadrant of the same eye. She complained of slight right-sided eyebrow ache, as well as generalized muscle pains and occasional paresthesias of the right hand. She had no history of collagen vascular disease.

Physical examination revealed stocking-glove hypalgesia on the right side for which no organic cause was found. Examination revealed a visual acuity of R.E.: 6/15 (20/50); L.E.: 6/6 (20/20). The right globe was tender and 2 mm proptotic. The eyes were white and not inflamed.

Binocular indirect ophthalmoscopy revealed a large inferonasal elevation of the right choroid with overlying retinal detachment. The surface of the subretinal elevated mass had a normal choroidal vascular pattern. Visual fields revealed a sloping field defect corresponding to the area of retinal and choroidal elevation. Because the normal choroidal vascular pattern was inconsistent with the diagnosis of intraocular melanoma made on the patient's ad-

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mission to the hospital, additional studies were done.

Neurologic consultation, electroencephalograms, and skull x-rays revealed no central nervous system abnormalities. A metastatic survey was negative. A ³²P uptake test of the ocular lesion done by current standard techniques showed positive findings of 186% at 24 hours and 140% at 48 hours. The patient was discharged without treatment for further observation

Three months later the patient developed severe pain in her right eye and was readmitted for further examination. The lesion in the right eye had increased significantly in size. A ³²P uptake test was again positive. A biopsy of the overlying conjunctiva showed only chronic inflammation. A-scan ultrasonography suggested a solid or multicystic mass at least 7 mm thick. Surgical exploration of the globe revealed a solid scleral mass that was causing a localized elevation on the outer surface of the eye. Biopsy specimens were taken from superficial areas as well as 8 mm into the mass.

Histologic examination of the specimens showed dense collagenous tissue diffusely infiltrated with numerous plasma cells and some lymphocytes. We found many fibroblasts and some epithelioid cells, but no organisms. A diagnosis of brawny scleritis was made.

The patient received systemic corticosteroids, which promptly alleviated the pain. The patient was given gradually tapered doses of corticosteroids for one year, and the lesion gradually diminished in size.

Three years later the patient was admitted for examination of 4 mm of proptosis of the right eye. Biopsy of a firm inner quadrant orbital mass showed inflammation similar to that noted on the previous scleral biopsy (Figs. 1 and 2). The inflammation again responded well to systemic corticosteroids.

Case 2—In July 1977, a 57-year-old white woman went to her ophthalmologist with a two-month history of a red left eye. The patient was in good health and had no history of collagen vascular disease. Her visual acuity was 6/6 (20/20) in both eyes, and the anterior segment was normal. Ophthalmoscopy showed an elevated lesion in the left eye at the 8 o'clock meridian, just in front of the equator. The findings on ultrasound corroborated the diagnosis of choroidal melanoma. The ³²P uptake test was 300% over the control. The left eye was enucleated.

Gross examination of the cut globe revealed a white scleral mass that elevated the underlying choroid (Fig. 3). The choroidal pattern appeared normal. Histologic examination was typical of brawny scleritis and showed mostly plasma cells and some lymphocytes infiltrating the markedly thickened sclera and the choroid. There was no evidence of scleral necrosis.

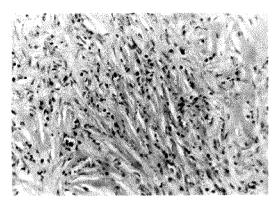


Fig. 1 (Feldon and associates). Case 1. Scleral biopsy shows diffuse infiltration by plasma cells and lymphocytes. The amount of collagen appears increased (hematoxylin and eosin, \times 250).

Case 3-A 70-year-old white man with clinically proven severe rheumatoid arthritis and suspected dermatomyositis went to his ophthalmologist in December 1976 with a four-week history of pain in the right eye. Six months previously an eye examination by the same physician revealed aphakia with visual acuity of 6/6 (20/20) in both eyes and no other abnormalities. Eve examination now revealed a best corrected visual acuity of R.E.: 6/60 (20/200); L.E.: 6/6 (20/20). The upper right evelid was slightly blepharoptotic, the conjunctiva mildly injected, and the anterior sclera severely thinned. Binocular indirect ophthalmoscopy of the right eye revealed a shifting retinal detachment overlying two large temporal choroidal masses. A diagnosis of choroidal melanoma was made, and a metastatic survey was negative. The right eye was enucleated.

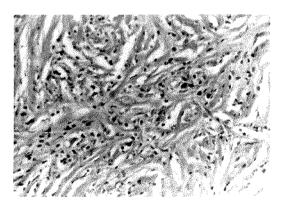


Fig. 2 (Feldon and associates). Case 1. Orbital biopsy three years after appearance of elevated subretinal lesion. Note the similarity of the infiltration by plasma cells and lymphocytes to the scleral biopsy shown in Figure 1. The mass consists of mainly fibrous connective tissue (hematoxylin and eosin, \times 250).

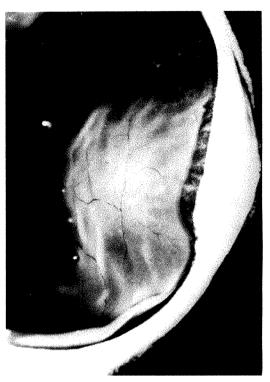


Fig. 3 (Feldon and associates). Case 2. Gross specimen enucleated for suspected melanoma. The mass consists of whitish tissue arising from the sclera. A normal choroidal vascular pattern is clearly visible overlying the lesion, especially near the cut edge of the specimen.

Histologic examination showed a mass arising in the sclera and extending from the temporal pars plana to immediately behind the equator (Fig. 4). Within the mass, the sclera was thickened and infiltrated with inflammatory cells, which destroyed the definition between coats of the eye. There were areas of necrosis and granulomatous reaction next to nodules of necrotic sclera. In other areas, lymphocytic and plasma cells infiltrated the sclera and adjacent choroid (Fig. 5). There was considerable choroidal pigment clumping and serous detachment of the retina.

Case 4—A 62-year-old white woman with a 24-year history of rheumatoid arthritis was admitted to the Massachusetts Eye and Ear Infirmary in March 1977 with a five-day history of progressive visual loss in the right eye and then in the left eye. Four weeks before admission, the patient had developed persistent eyebrow ache extending into the inferior orbital region. The patient had had uncomplicated bilateral cataract extractions ten years earlier, but had no other history of ocular, neurologic, or general physical disease. The symptoms developed despite long-term use of the anti-inflammatory agent ibuprofen (Motrin).



Fig. 4 (Feldon and associates). Case 3. Gross specimen enucleated for suspected melanoma. The mass appears to arise from the sclera, but distinct margins between choroid and sclera are absent. Focal necrosis can be seen within the tumor. Although sclera appears thickened in places, focal areas of thinning and necrosis are seen.

On physical examination, the patient had wasting and weakness of all extremities without myotonia or fasciculations. This was attributed to myositis or a secondary muscular dystrophy, or both. The etiology of an iron-deficient anemia could not be ascertained. A positive latex fixation test and the presence of antinuclear antibodies were reported. The Westergren sedimentation rate was 60 mm/hr.

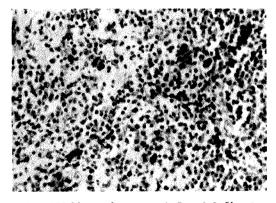


Fig. 5 (Feldon and associates). Case 3. Infiltration of sclera and choroid by lymphocytes and plasma cells is characteristic of scleritis (hematoxylin and eosin, \times 250).

On initial examination, visual acuity was R.E.: 6/12 (20/40); L.E.: finger counting at 1 foot. Peripheral constriction to confrontation field testing in both eyes, more marked inferiorly in the right eye and superiorly in the left eye, was sufficiently severe to prevent mobility without assistance. There was no proptosis. Trace conjunctival injection was present temporally in the left eye. The corneas were clear, and anterior chamber depth was consistent with surgical aphakia. Moderate cells and flare were observed in each eye, but the vitreous was not inflamed. Examination of the fundi revealed large choroidal detachments with overlying retinal detachments just sparing the right fovea and involving the entire left macula. Some vellow deposits appeared along the temporal arcades in the left eye. An ultrasound performed on the right eye showed large choroidal detachment with an expanded choroidal-scleral complex at the posterior pole consistent with inflammation or tumor." Edema of the subtenon's tissue was documented. Poor cooperation on the part of the patient precluded ultrasonography on the left eye.

During the patient's stay in the hospital, the inflammation and choroidal detachment of the left eye progressed. On the fourth day, we began giving the patient a daily dose of 60 mg of prednisone. Both eyes improved clinically. Four days after the initiation of therapy, the choroidal mound decreased, which left only minimal serous retinal elevation in the right eye. The left eye improved similarly after six days of therapy. Choroidal pigment lines marked the furthest extent of the choroidal detachment in

both eyes.

The patient was discharged with a tapering dose of corticosteroids. Repeat ultrasound of the right eye showed a substantial decrease in the posterior segment mass.

DISCUSSION

Scleritis is an uncommon disease, present in only 1/465 to 1/3000 of arthritis patients; of those, only 12 to 18% have posterior involvement.1 Although occasionally occurring in isolation, scleritis is often associated with collagen vascular diseases such as rheumatoid arthritis. periarteritis nodosum, Wegener's granrelapsing polychondritis, ulomatosis. or, occasionally, herpes zoster ophthalmicus.4,5

Symptoms may include severe boring pain, malaise, photophobia, and tearing. Anterior scleral nodules, proptosis, and keratitis may also be present.1 Histologically, scleritis characteristically appears as a nonpurulent central focus of scleral inflammation consisting primarily of plasma cells with a scattering of eosinophils. Surrounding the inflammation are epithelioid cells and Langhans' giant cells. The sclera may be destroyed and replaced by avascular fibrous tissue. Obliterative endarteritis is often seen.¹

Verhoeff and King⁶ differentiated between rheumatoid nodules of the sclera or scleromalacia perforans and brawny scleritis. They pointed out that brawny scleritis is more diffuse, has ill-defined boundaries, and has no tendency to form cavities or nodules. Verhoeff also noted that, in contrast to rheumatoid nodules which have necrosis and loss of tissue, brawny scleritis usually has a considerable increase in connective tissue with little necrosis. ^{6,7}

In our four cases of brawny scleritis, two resulted in enucleations for suspected melanomas, and in two, the corrected diagnoses were made and therapy initiated. The Table summarizes clinical, diagnostic, and histologic features of our cases and those from previous reports in which a clinical diagnosis of malignant melanoma was considered.^{2,3,7–11} These reports and others^{12–17} show that eyes with brawny scleritis are often enucleated after a misdiagnosis of melanoma. Verhoeff⁷ reported that a specimen was removed after the diagnosis of possible intraocular tumor.

Correlation of clinical and histopathologic findings alleviates some of the diagnostic confusion between melanoma and brawny scleritis, both of which appear ophthalmoscopically as elevated masses under the retina. In our first case, a normal choroidal vascular pattern was discerned clinically on the inner surface of the scleral mass. A normal choroidal pattern overlying the scleral mass appeared in the gross examination of the specimen in Case 2 (Fig. 3). Additionally, Tso¹⁰ presented a case of brawny scleritis; the eye, which on photography of the fundus revealed a normal choroidal vas-

cular pattern, was enucleated for suspected melanoma.

The absence of a normal choroidal vascular pattern, however, does not eliminate the possibility of brawny scleritis. Curtin8 studied a case in which the choroidal pattern was obscured by the retinal pigment epithelium except at the periphery of the tumor, where a normal vascular pattern appeared. Choroidal and retinal detachment may also obstruct viewing of the choroidal pattern, as in our Cases 3 and 4. Boylan, 11 Streeten, 9 Harper, 3 and Sears² presented similar cases. Detachment of choroid and retina may be a more common manifestation of brawny scleritis than a solitary elevated lesion. Changing the position of the patient to shift fluid away from areas of choroidal elevation may permit adequate viewing of the choroidal vascular pattern over the mass, which would facilitate the correct diagnosis. Also, the arcuate folds of the detached choroid may suggest choroidal effusion rather than choroidal tumor. Anterior signs of scleritis such as scleromalacia (as in Case 3) or scleral nodules may also aid in diagnosing posterior scleritis.

Serous choroidal and retinal detachment also appears in the uveal effusion syndrome.¹⁸ Differentiation of this condition from brawny scleritis can be difficult, especially if the scleritis is bilateral, as in our Case 4. However, uveal effusion usually occurs in males; there is no history of collagen vascular disease; the effusions and uveitis are not responsive to corticosteroids; and the cerebrospinal fluid protein is often increased.¹⁸ Also, ultrasound may show typical thickening of the sclera in scleritis.

In all of our cases as well as most of the previously reported cases of brawny scleritis, episodes of ocular pain, inflammation, or both, occurred sometime during their clinical course. Pain with melanoma or metastatic disease is uncommon in the absence of hemorrhage or secon-

CASES OF BRAWNY SCLERITIS CLINICALLY CONSIDERED TO BE CHOROIDAL MELANOMAS TABLE

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Reference	Age, Sex	Pain	Collagen Disease	32p Test	Ultrasound	Specimen	Fundus appearance
Present study Case 1*	33, F	+	=	+	-4-	Biopsy	Smooth elevated mass with normal
Case 2	57, F	+	I	+	**	Enucleated	Smooth electrical mass with normal
Case 3 Case 4	70, M 62, F	+ +	+ +		***	Enucleated	Choroidal and retinal detachments Choroidal and retinal detachments in both evest
Verhoeff ⁷	76, M	+	+			Enucleated	in composition of the following with shallow retinal detachment noted in
Boylan ¹¹	P, M	+	+			Enucleated	gross specimen Smooth contoured choroidal and
Harper³ Sears²	52, F 38, M*	++	1 1	+		Enucleated Biopsy	Massive seleral thickenings Rhegmatogenous retinal detachment over smooth elevated
Curtin ⁸	54, F		1		*	Enucleated	subretinal mass Focal mounds of pigment proliferation in choroid and retinal pigment epithelium with apparently normal choroidal pattern at edges
Streeten ⁹ Tso ¹⁰	74, F 53, F	+ t	+	+	ster ster	Enucleated Enucleated	of mass§ Choroidal and retinal detachment§ Smooth elevated mass with normal choroidal pattern‡

^{*}Scleritis suspected.

*Indicates scan consistent with diagnosis of choroidal melanoma.

†Fundus appearance determined by examination of the patient or by fundus photograph.

§Fundus appearance determined by examination or photograph of gross specimen.

dary glaucoma; uveal effusion syndrome, however, may be painful.

Proptosis and diplopia occasionally occur in brawny scleritis because of the associated inflammation within the orbit,⁵ which, in Case 1, became manifest several years after the initial biopsy diagnosis of brawny scleritis. Biopsy showed inflammation in the orbit similar to that in the sclera (Figs. 1 and 2).

Diagnostic tests such as 32P uptake and ultrasonography can be misleading. The ³²P test was always positive and the ultrasound always consistent with melanoma when performed on our scleritis patients and on those of others referred to in the Table. The ³²P uptake that can be positive in a variety of inflammatory, vascular, hemorrhagic, osseous, and cancerous conditions of the posterior segment. 14,19 Ultrasound examination revealed melanoma in three of our four cases and in the three other cases referred to in the Table. Ultrasound cannot always resolve adjacent tissues in the posterior segment sufficiently to allow definitive diagnosis. Although choroidal detachment may show a sonic clear space between retina and sclera, both scleritis and melanoma may appear as solid tumors with echo decay. shadow effect, and acoustic quiet zone.20 These tests may make the unsuspecting surgeon comfortable with the erroneous diagnosis of melanoma, despite clinical evidence to the contrary.

If after clinical and diagnostic examination, the diagnosis is still doubtful, the globe may be explored. In Case 1, the increased scleral bulk produced an area of whitish elevation on the surface of the globe similar to that described by Verhoeff.⁶ The biopsy of the lesion in our case was performed without complication to a depth of 8 mm, which confirmed the diagnosis of brawny scleritis. Sears² performed a similar biopsy while buckling a coexisting rhegmatogenous retinal detachment in the same eye.

Systemic corticosteroids are useful in the treatment of scleritis.^{21,22} Corticosteroids successfully controlled the inflammation and resulted in clinical resolution of the posterior segment findings in Cases 1 and 3. Others, however, have suggested that immunosuppressive and anti-inflammatory agents be used with or instead of corticosteroids.^{4,23} One of our patients (Case 4) was taking ibuprofen when bilateral scleritis developed; yet she responded quickly to moderate doses of systemic prednisone.

SUMMARY

We studied four patients with posterior brawny scleritis. Two underwent enucleation for suspected melanoma, and in the other two, the correct diagnosis was made and effective therapy begun. Of seven other eyes with brawny scleritis from other sources, five were enucleated after diagnosis of choroidal melanoma and one for suspected intraocular tumor. This experience and other previous reports indicate the high incidence of diagnostic confusion regarding brawny scleritis. We therefore emphasized clinical symptoms and signs of brawny scleritis: inflammation, tenderness or pain of the globe. history of collagen vascular disease, proptosis, bilaterality, and retinal and choroidal detachment. A preserved normal choroidal vascular pattern over an elevated subretinal mass may be indicative of posterior brawny scleritis. Scleral biopsy is useful for tissue diagnosis. Radioactive phosphorus uptake tests and ultrasonography may erroneously indicate choroidal melanoma and lead to enucleation of a potentially salvageable globe.

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CELLULAR IMMUNITY IN MOOREN'S ULCER

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Mooren's ulcer is a chronic, painful ulceration of the cornea that begins in the periphery with an undermined and occasionally infiltrated leading edge. The ulcer advances centripetally and circumferentially leaving in its wake a thin, vascularized cornea. An unequivocal cause has not been established for this often relentlessly progressive condition. In recent years, an autoimmune basis has been suggested for Mooren's ulcer. In 1969, circulating antibodies to corneal epithelium were demonstrated in a patient with Mooren's ulcer, but immunoglobulins bound to the patient's own cornea could not be found. The finding in another report that the subepithelial tissues of the conjunctiva were packed with plasma cells provided further support for an antibody-dependent disease.² Recent reports have demonstrated that immunoglobulins and complement bound to the conjunctival epithelium and circulating antibodies to conjunctival and corneal epithelium are a consistent finding in Mooren's ulcer.^{3,4} Whether these humoral autoimmune phenomena are the cause or consequence of Mooren's ulcer is speculative.

Cell-mediated autoimmunity has been reported in chronic diseases of the cornea by using the leukocyte migration inhibition test.⁵ To date, no such cellular immunity studies have been reported for patients with Mooren's ulcer. Our purpose

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in this report is to demonstrate that cellular as well as humoral autoimmune phenomena are found in Mooren's ulcer. We studied the lymphocytes of seven patients with Mooren's ulcer for evidence of macrophage migration inhibition factor production in the presence of corneal antigen.

SUBJECTS AND METHODS

We studied seven patients (Table) with the clinical appearance of Mooren's ulcer: a painful, peripheral ulceration of the cornea with an undermined edge advancing both centripetally and circumferentially and leaving behind a thinned vascularized cornea. All patients, except in Case 2, underwent macrophage migration inhibition factor testing when the disease was inactive.

The corneal stroma of both eyes of Cases 6 and 7 had been totally destroyed, thus leaving only Descemet's membrane covered by vascularized epithelium. Thereafter, disease activity subsided, and both patients underwent unilateral penetrating keratoplasties and lens extractions. After corneal grafting, the patients received hourly topical 0.1% dexamethasone, as well as occasional subtenon's injections of 40 mg of triamcinolone acetate for several months. Case 6 underwent macrophage migration inhibition factor testing ten months postoperatively, and Case 7, five months postoperatively. When undergoing macrophage migration inhibition factor testing, Case 6 was receiving topical fluorometholone hourly, and Case 7, six times per day. Both patients were studied twice, and Case 6 was tested on one occasion during an episode of graft rejection.

Controls consisted of seven normal individuals.

TABLE

MIGRATION INHIBITION INDICES IN PATIENTS WITH MOOREN'S ULCER

Case Sex, Age No., (yrs)	Duration of Disease (yrs)	Laterality	Extent of Corneal Involvement	Status When Studied	Migration Inhibition Index*
1, M, 62	3.5	Bilateral	2 quadrants, right eye 1 quadrant, left eye	Inactive	0.49
2, M, 23	0.75	Unilateral	2 quadrants, right eye	Active	0.37
3, M, 68	3	Bilateral	Total, right eye 2 quadrants, left eye	Inactive	0.51
4, F, 64	1	Unilateral	1 quadrant, left eye	Inactive	0.72
5, M, 57	2	Unilateral	1 quadrant, right eye	Inactive	0.40
6, F, 74	3.5	Bilateral	Total, both eyes	Inactive Graft rejection episode	1.23 0.30
7, F, 46	2.5	Bilateral	Total, both eyes	Inactive Inactive	0.82 0.96

^{*}A value <.75 demonstrates positive migration inhibition in response to corneal antigen.

Antigen preparation—Donor eyes from patients with O blood type were obtained within 12 hours of donor death. The corneas were removed and pooled. The corneas were minced with scissors and then homogenized at O° C in phosphate-buffered saline at pH 7.4 in a homogenizer. The homogenate was stirred at 4° C overnight and then centrifuged at 30,000 r.p.m. for 30 minutes. The supernatant was removed and dialyzed against phosphate-buffered saline for 36 to 48 hours. The extracts were sterilized by Millipore filtration (0.22 μ filters). Protein determination was performed and the extract was adjusted to a concentration of 5 mg/ml.6 At the time of each macrophage migration inhibition procedure, aliquots were prepared at concentrations of 1:5, 1:10, and 1:20 by using tissue culture medium 199. These concentrations did not show a nonspecific cytotoxicity or inhibit the migration of guinea pig macrophages.

Lymphocyte separation—Whole blood (30 ml) was collected in plastic syringes

containing preservative-free heparin (1,000 units of heparin per 30 ml of blood). The mononuclear cells were collected by centrifugation by previously published methods.⁷ The mononuclear cells were washed three times in tissue culture medium 199 and were composed of 80 to 95% lymphocytes.

Migration inhibition test-Guinea pig macrophages were prepared and collected according to previously published methods. 8 The guinea pig macrophages were counted, and mononuclear cells from either patients or normal controls were added to a volume of 20% of the total cell suspension. Capillary tubes were filled with the cell suspension and centrifuged at 500 r.p.m. for five minutes. The capillary tubes were then cut at the cell-fluid interface. The capillary tubes containing the cells were placed in Lab-Tek slides divided into four separate chambers. To each of the chambers, we added 0.8 ml of tissue culture medium 199 containing 15% heat-inactivated fetal calf serum. Four capillary tubes in two separate chambers were run without antigen as controls. At least four capillary tubes in two separate chambers were used for each dilution of corneal antigen (1:5, 1:10, 1:20). To these chambers, 0.2 ml of antigen was added in the appropriate dilution. An additional control included with each experiment involved capillary tubes filled with macrophages without added lymphocytes, with and without antigen in the chamber. The chambers were incubated for 18 to 24 hours at 37° C.

We determined the area of migration by projecting and tracing the image of cellular migration within each chamber on a sheet of paper. The images were cut out and weighed. The mean of the four weights for each dilution of corneal antigen was obtained. The migration index was calculated by dividing the average weight of migration fan cutouts with antigen by the average weight of migration fan cutouts without antigen. By testing the patient's lymphocytes against different concentrations of corneal antigen, we obtained a dose-response curve; this permitted determination of the antigen concentration which caused the maximal macrophage migration inhibition value for each patient's lymphocytes. Cases 1, 2, 3, and 6 demonstrated maximal migration inhibition at a 1:10 dilution of corneal antigen, while Cases 4, 5, and 7 showed maximal inhibition at a 1:20 dilution. The maximal macrophage migration inhibition value for each patient is recorded in the Table. A value <.75 is considered evidence of positive migration inhibition.

RESULTS

All patients except one (Case 7) demonstrated positive macrophage migration inhibition in response to corneal antigen. Results were negative in Case 6 on one occasion, but became positive during an episode of graft rejection. Seven normal individuals who were test-

ed in conjunction with the patients did not demonstrate macrophage migration inhibition in response to corneal antigen.

DISCUSSION

Lymphocyte production of macrophage migration inhibition factor in response to an antigen indicates that the lymphocytes are sensitized to that particular antigen.9 The results of this study demonstrate that lymphocytes from six of seven patients with Mooren's ulcer were sensitized to a saline soluble corneal antigen. Cellmediated immune reactions which are described in association with Mooren's ulcer in this report are not unique to this condition, but are found with other chronic diseases of the cornea.⁵ The significance of these findings is speculative. Nevertheless, cell-mediated as well as humoral autoimmunity may play a role in the cause of Mooren's ulcer.

All patients in this series demonstrated positive macrophage migration inhibition factor production in response to corneal antigen on initial testing, except those in Cases 6 and 7. These latter patients had the only cases of total corneal stromal destruction. When disease activity had subsided for several months, both these patients underwent unilateral penetrating keratoplasties and lens extractions. Case 6 underwent macrophage migration inhibition factor testing ten months after corneal grafting, and Case 7, five months after corneal grafting. The negative migration inhibition factor test in these two patients may be explained in several ways. First, the patients may not have been sensitized to corneal antigen and cell-mediated immunity may not be an integral part of this disease. It is also possible, however, that bilateral corneal destruction in these two patients reduced the volume of corneal antigen below that required to maintain lymphocyte sensitivity. Lastly, these two patients had been receiving intensive topical and subtenon's corticosteroid therapy for their corneal grafts, and systemic absorption of corticosteroids¹⁰ may have altered the lymphocytic response to corneal antigen in the macrophage migration inhibition factor test or caused sensitized lymphocytes to leave the circulation sequestered in lymph nodes.

The patient in Case 6 was retested when undergoing a homograft rejection in the right eye. At this time, she demonstrated positive macrophage migration inhibition factor production. The demonstration of positive and negative reactions in the macrophage migration inhibition factor assay in the same patient at different times has been previously reported. Fluctuations in lymphokine production could be involved, or the positive response in this case may have been caused by a homograft rejection. 13

SUMMARY

Seven patients with the clinical diagnosis of Mooren's ulcer underwent macrophage migration inhibition factor testing for evidence of cellular immunity to corneal antigen. Six of seven patients demonstrated positive macrophage migration inhibition in response to corneal antigen, thus suggesting that cell-mediated immunity may play a role in the cause of Mooren's ulcer. One of these patients had a negative macrophage migration inhibition factor when the disease was inactive, but developed a positive response in association with a homograft rejection episode.

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EXPERIMENTAL AND CLINICAL DATA ON THE INSERTION OF THE LEVATOR PALPEBRAE SUPERIORIS MUSCLE

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The etiology of the upper eyelid skin crease1.p.117 and the functional importance of the insertions of the levator palpebrae superioris muscle have been disputed.² Whitnall³ stated that the skin crease lies 2 to 3 mm above the highest point of the cutaneous insertion of the levator palpebrae superioris muscle and it is the contraction of this muscle that causes the crease to become deeply recessed. This has been accepted by most authors.4-8 Isaksson,1 however, pointed out that all the palpebral skin is freely mobile over the underlying orbicularis muscle and that when an upper eyelid becomes edematous, or local anesthetic is injected subcutaneously, the skin crease disappears. This could be explained by relaxation of the levator palpebrae superioris muscle as occurs during sleep. Werb² believes that the lower border of the aponeurosis is free. If this is true, then disinserting the aponeurosis should not result in blepharoptosis and one might expect changes in Müller's muscle or its insertion in clinical cases of blepharoptosis. We have tried to assess these possibilities.

SUBJECTS AND METHODS

Experiments were performed on monkeys since the eyelid anatomy of these

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primates is the most similar experimental model to human evelid anatomy available. Eight rhesus monkeys were anesthesized with intramuscular ketamine (20 to 30 mg/kg of body weight), which was supplemented by injections of local anesthetic. A skin incision was made under the brow and deepened through the orbicularis muscle to expose the orbital septum which was divided. Wire markers were sutured to the aponeurosis at its iunction with the levator palpebrae superioris muscle and to the periosteum of the orbital rim. The skin and orbicularis muscle were then closed. A marker was sutured into an epidermal scratch incision in the skin crease. A fourth marker was placed in the upper border of the tarsal plate by a posterior approach. The eyelids were sutured together with a central tarsorrhaphy. The eyelid skin was then ballooned forward and separated from the underlying orbicularis by a subcutaneous injection of saline. Radiographs were taken before and after the injection (Figs. 1 and 2). The tarsorrhaphy suture was then cut. When the saline had dispersed, a series of radiographs were taken to get views of the eyelids fully open and in varying degrees of voluntary closure. The animals were awake but sedated with kitamine.

A skin crease incision was made in three rhesus monkeys and deepened to expose the aponeurosis. This was disinserted from the anterior surface of the tarsus and the orbicularis muscle (Fig. 3). Part of the aponeurosis was resected but Müller's muscle was left undisturbed. The skin and orbicularis muscle were then closed.

Müller's muscle was excised in three

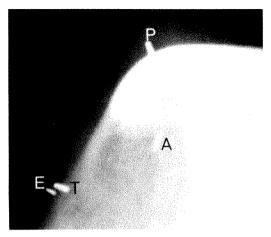


Fig. 1 (Collin, Beard, and Wood). Radiograph of monkey skull showing markers in brow periosteum (P), aponeurosis (A), upper border of tarsus (T), and epidermis of skin crease (E).

rhesus monkeys by a posterior approachwhich left the aponeurosis intact. The conjunctiva was sutured to the upper border of the tarsus.

Six human and six rhesus monkey eyelids were dissected, and sections were compared by light microscopy (Figs. 4 and 5). Electron microscopic studies were carried out on the monkey eyelids. The

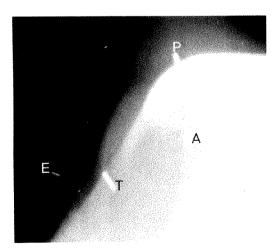


Fig. 2 (Collin, Beard, and Wood). Radiograph of monkey skull after injection of saline between the skin and orbicularis muscle showing separation of epidermal marker (E) and tarsal marker (T) but no movement of the aponeurosis (A).

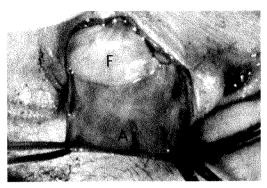


Fig. 3 (Collin, Beard, and Wood). Monkey levator aponeurosis (A) being disinserted showing preaponeurotic fat pad (F).

tissues were fixed for transmission electron microscopy with 3% paraformaldehyde-glutaraldehyde in 0.2M sodium cacodylate buffer with the addition of 1% sucrose, 0.2% calcium chloride and potassium chloride for several hours at 20° C. The tissues were then washed in 0.2M sodium cacodylate and 0.5M sucrose and postfixed with 2% osmium tetroxide in sodium veronal acetate buffer, and stained en bloc with Kellenberger uranyl acetate stain for three hours. The tissues were rinsed after the staining period, dehydrated in graded alcohol 70% to 100%, then propylene oxide, and finally embedded in Araldite 502. Ultrathin sections for

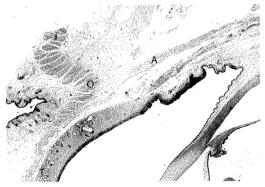


Fig. 4 (Collin, Beard, and Wood). Histologic section of human upper eyelid showing aponeurosis (A) inserting into septa between orbicularis muscle bundles (O) and onto tarsus (T) (hematoxylin and eosin, ×10).



Fig. 5 (Collin, Beard, and Wood). Histologic section of monkey upper eyelid showing aponeurosis (A) inserting into septa between orbicularis muscle bundles (O) and onto tarsus (T) (Masson's trichrome, ×40).

electron microscopy were mounted on grids, stained primarily with 2% uranyl acetate followed by lead citrate. They were examined and photographed with an electron microscope.

Histologic sections of the upper border of the tarsus and Müller's muscle were examined by light microscopy from 20 human patients operated on for blepharoptosis. These included four cases of congenital blepharoptosis and three cases clinically diagnosed as having a disinsertion of the aponeurosis of the levator palpebrae superioris muscle.9 The specimens were removed either as part of the blepharoptosis procedure as with a Fasanella-Servat operation, 10 or were taken as a separate biopsy specimen as in the repair of an aponeurosis disinsertion. These were compared with specimens taken by similar operative techniques from six cadavers with ages ranging from a stillborn fetus to 75 years old, and with 14 exenteration specimens. The specimens were embedded in paraffin and sections stained with hematoxylin and eosin and Masson's trichrome stain.

RESULTS

When the skin and orbicularis muscle of a monkey were separated with an injec-

tion of saline, there was no movement of the marker in the levator aponeurosis (Figs. 1 and 2). However, it moved the same distance that the markers in the epidermis and upper border of the tarsal plate moved when the eye opened and closed. This confirmed that the markers had been placed in their intended positions. When the saline dispersed, the skin crease reformed exactly at its original position. If there was a direct skin insertion of the collagen fibers from the aponeurosis and the levator muscle did not relax, the fibers would have ruptured since collagen is poorly elastic and could not have stretched sufficiently. If the fibers had ruptured, then it would be difficult to explain how the skin crease reformed in exactly the same place. The most likely explanation is that it is the insertion of the aponeurosis to the septa between the orbicularis muscle bundles which governs the skin crease and that there is no direct insertion into the skin.

A blepharoptosis developed postoperatively in the monkeys that had a disinsertion of the aponeurosis (Fig. 6) and in those that had a Müllerectomy. In both groups the blepharoptosis healed itself after about two weeks (Fig. 7). If the aponeurosis and Müller's muscle were both excised, a permanent blepharoptosis resulted. This shows that in a monkey

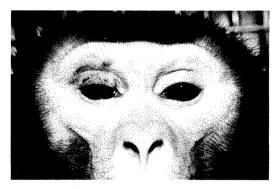


Fig. 6 (Collin, Beard, and Wood). Monkey, two days post disinsertion of the right levator aponeurosis showing right blepharoptosis.

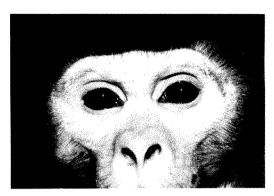


Fig. 7 (Collin, Beard, and Wood). Same monkey two weeks after disinsertion of the right levator aponeurosis.

both the aponeurosis and Müller's muscle normally contribute to eyelid elevation. If one is cut, organization and fibrosis will rapidly restore the eyelid level provided the other is intact. If both are cut, a permanent blepharoptosis results.

The dissections and light microscopy of the upper eyelids in man and the rhesus monkey confirmed the similarity between the two (Figs. 4 and 5). In both, the aponeurosis was found to insert between the orbicularis muscle bundles beginning approximately at the level of the upper border of the tarsus in the area of the skin crease and onto the lower half of the anterior surface of the tarsus. Electron microscopy demonstrated that in a monkey the aponeurosis consists of regularly arranged collagen (Fig. 8), which is continuous with the septa between the orbicularis muscle bundles (Fig. 9). There was no specific attachment of the collagen fibers to the skin in the skin crease area and these fibers were equally haphazard in the pretarsal, preseptal, and skin crease areas (Fig. 10). These findings are similar to the light microscopy and electron microscopy¹¹ reports in man, and the similarity between the human and monkey evelid microanatomy has previously been described.11

We measured the length of Müller's

muscle tendon in 20 cases of blepharoptosis and 20 controls by light microscopy and found that it varied from 0.5 to 1.5 mm with an average of about 1 mm. With increasing age there is a tendency for Müller's muscle and its tendon to become more tenuous and for increasing fat deposits to occur in the upper border of the tarsus (Figs. 11 and 12). This tendency, however, was no more marked in the cases of blepharoptosis than in the control specimens. We did not therefore find any evidence of a disinsertion of Müller's muscle. The length of the tendon is difficult to measure accurately as the collagen of the tendon is continuous with the collagen of the tarsus itself (Fig. 13). There are obvious inaccuracies in any measurements made on small biopsy specimens and in particular after the tissues have been crushed with hemostats as with a Fasanella-Servat procedure. 10 In both the controls and the blepharoptosis specimens Müller's muscle only averaged 0.1 to 0.5 mm in thickness. There was a layer of vascular connective tissue between the conjunctival epithelium and Müller's muscle which varied between 0.1 and 1.00 mm in thickness and there was another vascular laver between Müller's muscle and the aponeurosis that contained the peripheral marginal arcade of vessels. This means that the surgeon operating on Müller's muscle is holding mainly vascular connective tissue and only a thin strip of actual muscle.

DISCUSSION

Berke and Wadsworth⁶ found that Müller's muscle was always present in congenital blepharoptosis. Putterman and Urist¹² described a Müller's muscle-conjunctiva resection procedure. Their histology in 25 patients operated on for both congenital and acquired blepharoptosis confirmed that Müller's muscle was present in all their specimens. Kuwabara, Cogan, and Johnson¹¹ did not find any

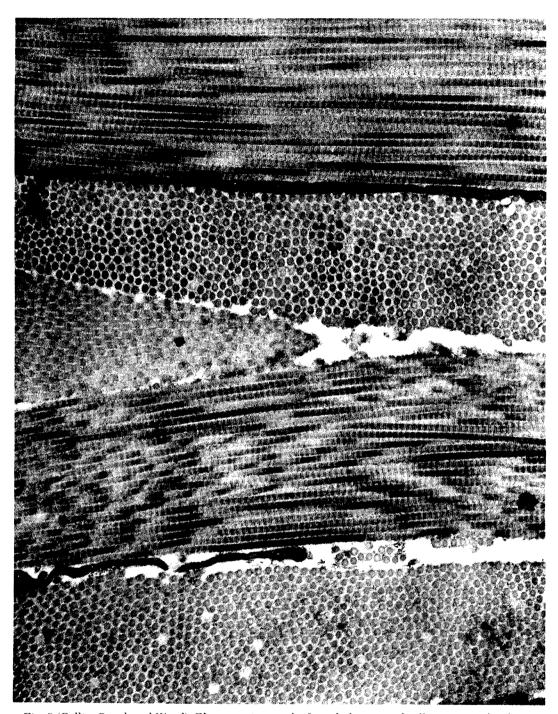


Fig. 8 (Collin, Beard, and Wood). Electron micrograph of regularly arranged collagen in monkey levator aponeurosis (\times 17,550).



Fig. 9 (Collin, Beard, and Wood). Electron micrograph of regularly arranged collagen of the monkey aponeurosis (A) inserting between the orbicularis muscle bundles (O) (\times 2,670).



Fig. 10 (Collin, Beard, and Wood). Electron micrograph of irregularly arranged collagen in area of skin crease between skin and orbicularis in a monkey (\times 11,700).

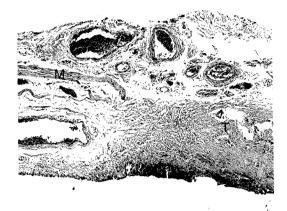


Fig. 11 (Collin, Beard, and Wood). Specimen removed by Fasanella-Servat procedure from a 6-year-old patient with congenital blepharoptosis showing the short distance between Müller's muscle (M) and tarsus (T) (hematoxylin and eosin, \times 80).

anatomical abnormality electron microscopically in Müller's muscle in a series of both congenital and acquired blepharoptosis. We did not find any evidence for a disinsertion of Müller's muscle in the cases of congenital and acquired blepharoptosis we examined. However, we did find that Müller's muscle and its tendon normally became thinner and elongated



Fig. 12 (Collin, Beard, and Wood). Specimen removed by Fasanella-Servat procedure from a clinically diagnosed case of aponeurosis disinsertion aged 75 showing Müller's muscle (M), a longer tendon (B) than in Fig. 11, and increased fat (F) in the top of the tarsus (T) (hematoxylin and eosin \times 80).

with increasing age. The available studies therefore have not found any specific defects of Müller's muscle in cases of blepharoptosis. However, many cases of acquired blepharoptosis have been shown to be associated with defects of the aponeurosis. This suggests that the normal aponeurosis is at least partly responsible for eyelid elevation. When Müller's muscle is paralyzed in Horner's syndrome, there is approximately 2 mm of blepha-



Fig. 13 (Collin, Beard, and Wood). Electron micrograph of regularly arranged collagen in monkey Müller's muscle tendon (B) merging into the irregularly arranged collagen of the tarsus (T) (× 1,520).

roptosis. Many eyelids in acquired blepharoptosis are lower than this, which supports the concept of an aponeurotic defect if the levator muscle is normal.

Henderson¹⁴ described a disinsertion of Müller's muscle for the relief of evelid retraction. If a greater correction was required, he excised the insertion of the aponeurosis into the anterior surface of the tarsus. Putterman and Urist¹⁵ excise Müller's muscle and selectively disinsert part of the aponeurosis until the required degree of eyelid drop is achieved. In our experiments with monkeys either disinserting the aponeurosis or doing a Müllerectomy caused a blepharoptosis. Organization of the wound in monkeys is rapid and the blepharoptosis soon cured itself. Such organization also occurs in humans. We have found that after any surgery for the relief of eyelid retraction, the effect is enhanced if the eyelid is put on traction with a suture taped to the cheek. Massage of the eyelid subsequently helps to prevent the evelid reverting to its original level as the wound contracts. Lauring16 described a sutureless Fasanella-Servat operation in which part of the tarsus and the lower part of Müller's muscle is excised. The cut edges heal by organization without being held together with a suture. He suggests that the pull of the levator muscle via the insertion of the aponeurosis into the lower third of the tarsus acts as a biological suture. If the aponeurosis does not contribute to eyelid elevation this procedure should produce and not cure a blepharoptosis. The available evidence therefore strongly suggests that the aponeurosis and Müller's muscle work synergistically to raise the eyelid. We cannot therefore agree with Werb² that the lower border of the aponeurosis is normally free.

If the eyelid crease is made by the attachment of the aponeurosis to the septa

between the orbicularis muscle bundles and not directly to the skin itself, it is much easier to explain the high evelid fold associated with a disinsertion of the levator aponeurosis. When the terminal part of the aponeurosis stretches or disinserts, the levator muscle retracts and pulls the orbital septum with it. The orbicularis is firmly bound to the septum^{3, p.125} and it too is pulled backwards. The evelid skin follows the orbicularis and the evelid fold becomes higher as more septum is pulled back into the orbit. This is enhanced by atrophy of a pre-aponeurotic fat pad and there is no need to postulate stretching of the cutaneous insertion of the aponeurosis.

SUMMARY

Radiographic and electron microscopic evidence showed that the upper evelid skin crease is formed by the insertion of the levator palpebrae superioris muscle into the septa between the orbicularis muscle into the septa between the orbicularis muscle bundles and not into the skin itself. Experiments on monkeys showed that the insertions of the aponeurosis and of Müller's muscle both contribute to normal eyelid elevation. No histologic evidence was found for a disinsertion of Müller's muscle in 20 cases of blepharoptosis. This, with other evidence discussed, supports the functional importance of the human aponeurotic insertions in eyelid elevation.

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OPHTHALMIC MINIATURE

The flying snoll—a lizard with four hundred eyes, two hundred for distance and two hundred for reading. According to legend, if a man gazes directly into the face of the snoll he immediately loses his right to drive in New Jersey.

Woody Allen, Without Feathers New York, Random House, 1975

A MODIFIED SILICONE FRONTALIS SLING FOR THE CORRECTION OF BLEPHAROPTOSIS

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A frontalis sling is usually indicated in cases of blepharoptosis with minimal or no levator function. Various materials have been used for the sling, including autogenous skin, ¹⁻³ nonabsorbable suture, ⁴ preserved and autogenous fascia, ⁵ collagen tape, ⁶ and silicone. ^{7,8} This report describes a modification of the technique described by Tillett and Tillett, ⁷ which uses the No. 40 silicone band.

Метнор

The operation can be performed under local or general anesthesia. If the anesthesia is local, a frontal nerve block9 or local infiltration, or a combination of both, is used. The following technique is used for the frontal nerve block: a 4-cm, No. 25 needle is passed under the superior orbital rim at the midorbital point and is advanced posteriorly hugging the roof of the orbit until the hub of the needle is reached. The tip of the needle should be near the superior orbital fissure where the frontal and lacrimal nerves, which give most of the sensory supply to the upper eyelid and forehead, enter the orbit. An injection of 0.75 ml of 2% lidocaine with epinephrine is made and can be augmented by local infiltration if necessary.

Two incisions 6 mm long are made 5 mm superior to the eyelash line in the middle and lateral one third of the upper eyelid (Fig. 1, top left). The tarsus is exposed, and a double armed 4-0 dacron suture is vertically placed deep in the tarsus through each incision. Above the eyebrow line, incisions exposing the deep

frontalis fascia are made centrally, medially, and laterally. A Wright fascia needle is then placed through the medial eyebrow incision and carefully advanced over the superior orbital rim inferiorly and posteriorly to approximate the level of the levator aponeurosis. The needle is then directed anteriorly in the evelid to emerge in the medial eyelid incision at the level of the tarsus. A No. 40 silicone band is threaded through the eye of the fascia needle and brought back through the eyelid and out the eyebrow incision. The 4-0 dacron suture is then passed through the end of the silicone band in a vertical mattress fashion and tied, thus securing the band to the tarsus. An additional suture can be placed to stabilize the band further. A second silicone band is passed through the lateral brow and evelid incisions in a similar manner.

Through the medial and lateral evebrow incisions, a suture of 4-0 Prolene is passed through the deep frontalis fascia and tied loosely to act as a pulley or guide for the silicone band (Fig. 1, top right). The fascia needle is then passed through the central eyebrow incision to the medial and then to the lateral eyebrow incisions to bring both ends of the silicone bands out the central incision. The surgeon adjusts the silicone bands to achieve the desired eyelid level, which is usually at the superior corneoscleral limbus, while the assistant ties a doublethrow loop of suture or a clove hitch to secure the overlapping bands. Before the tying of the suture, the eyelid is forcibly pulled down to make certain the bands are tight within the tracks. Tension on the bands is also adjusted to achieve the desired evelid contour. A double armed 4-0 dacron is then placed through the bands so that

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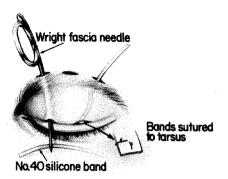
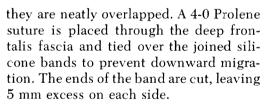
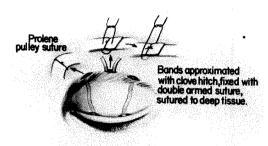


Fig. 1 (Leone and Rylander). Top left, Incisions are made 5 mm from the eyelash line thus exposing the tarsus medially and laterally. At the top of the eyebrow, incisions are made medially, centrally, and laterally. A Wright fascia needle is threaded through the medial and lateral evebrow incisions to the level of the superior orbital rim; from there, it is directed posteriorly to approximately the level of the levator aponeurosis. The needle is then directed forward to emerge at the level of the tarsus. The bands are secured to the tarsus with a double armed 4-0 dacron with an additional one placed for further support. Top right, 4-0 Prolene sutures, loosely tied through the medial and lateral evebrow incisions, act as pulleys for the bands. The two bands are joined in the central eyebrow incision with a clove hitch or double-throw suture when the desired eyelid level is obtained. The overlapped bands are then tied with a double armed 4-0 dacron and stabilized to the deep tissue with 4-0 Prolene. Bottom right, The subcutaneous tissue is sutured with 6-0 plain catgut and the skin with 6-0 silk.



If there is skin redundancy, an incision is made in the eyelid fold exposing the anterior surface of the tarsus and the appropriate amount of skin is excised.

The incisions are closed with 6-0 plain catgut in the subcutaneous tissue, and 6-0 silk in the skin (Fig. 1, bottom right). Antibiotic ointment is placed liberally over the palpebral fissure, and wet eye pads are loosely applied. The eye pads are kept in place except during meals for the first 24 hours.





If a reoperation is necessary for underor overcorrection, a small amount of local anesthetic is placed in the central eyebrow area, and an incision is made exposing the overlapped silicone bands. The two bands are untied, and the desired level is obtained by either loosening or tightening the bands to complete the procedure as previously described.

CASE REPORTS

Case 1—A 50-year-old black woman (Fig. 2) had chronic progressive external ophthalmoplegia with no levator function; she had bilateral silicone frontalis slings in March, 1970. Her upper eyelid level has remained constant in the resting state and varies with the use of her frontalis muscle. With forced closure, she is able to close her fissure. Although she has no Bell's phenomenon, she has shown only minimal signs of exposure keratopathy.





Fig. 2 (Leone and Rylander). Left, Preoperative appearance of a 50-year-old black woman with chronic progressive external ophthalmoplegia and complete blepharoptosis. Right, Postoperative appearance seven years after bilateral silicone frontalis slings.

Case 2—A 74-year-old white man (Fig. 3) had chronic progressive external opthalmoplegia and complete blepharoptosis. A bilateral frontalis sling with silicone bands was done in December, 1976; this resulted in an improvement of his upper eyelid position, but this was only partially satisfactory to the patient. A tightening of the band through the central eyebrow incision, done in March 1977, resulted in an improved upper eyelid position.

RESULTS

Of 22 frontalis slings, one became infected and partially extruded, but was

corrected by repositioning the bands. The longest follow-up has been seven years without complication. One band separated from the tarsus because of a superficial suture placement. This separation required insertion of a new band, which was tied with the opposite band in the central eyebrow area. There have been no overcorrections, but seven eyelids underwent repositioning through the central eyebrow incision for undercorrection.



Fig. 3 (Leone and Rylander). Top left, Preoperative appearance of a 74-year-old white man with chronic progressive external ophthalmoplegia and complete blepharoptosis. Top right, Undercorrected appearance after bilateral silicone frontalis slings. Bottom left, Postoperative appearance six months after revision of the silicone frontalis slings through the central eyebrow incision. Bottom right, Attempted closure shows good approximation of the eyelids.

DISCUSSION

In the original description by Tillett and Tillett,⁷ the eyelid was suspended by using a continuous silicone band with a loop running anterior to the tarsus. In our modification, the ends of each band are sutured to the tarsus to avoid the bulk of the extra loop and prevent migration. The side-by-side, overlapped tie of the bands at the central eyebrow incision avoids a rabbit-ears effect and allows 0.5 cm on each side of the tie. This excess silicone permits relaxation of the suspension should exposure or asymmetry occur.

The two Prolene "pulley" sutures provide a broad base for the silicone sling in the frontalis muscle. This broad base minimizes downward migration and relaxation of the suspension. The pulley sutures also permit the eyelid position to be adjusted through the central eyebrow incision only. Any revision does not involve opening the skin over the pulley sutures.

A frontal nerve block offers the advantage of giving sensory anesthesia to the upper eyelid and forehead area while preserving motor function, which allows the patient to close his eyes while the band is being adjusted. Additionally, the tissue is not distorted or ballooned by local infiltration.

Although most oculoplastic surgeons prefer autogenous or perserved fascia lata, we do not recommend that this method supplant the use of fascia lata, but that it be used in selected cases, particularly those with acquired blepharoptosis with ophthalmoplegia. The elastic quality of the silicone bands allows almost complete approximation of the eyelids with forced closure which helps to minimize exposure keratopathy. Moveover, older patients usually have decreased tear secretion which would make it more desirable to achieve a slightly undercorrected eyelid position to avoid symptoms of ex-

posure keratopathy. This would also be important in patients with chronic progressive ophthalmoplegia where a Bell's phenomenon may be impaired. Because silicone is nonbiodegradable, and is usually surrounded with a connective tissue envelope, readjustment of the eyelid position can be accomplished months or years after the initial surgery through a small central brow incision.

SUMMARY

We devised a frontalis sling by using two No. 40 silicone bands. Each free end was sutured to the tarsus and joined in the central brow area. We used pulley sutures in the medial and lateral brow incisions to prevent migration of the bands. With this method, adjustment of the evelid level can be made anytime postoperatively through the central eyebrow incision because of the ease in finding the overlapped ends of the silicone bands. This is particularly useful for patients with progressive ophthalmoplegia where poor closure and exposure keratopathy are potential postoperative problems.

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OPHTHALMIC SEQUELAE OF INFANTILE HEMANGIOMAS OF THE EYELIDS AND ORBIT

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"Infantile" or "strawberry-type" hemangioma of the eyelids is a nonheritable benign tumor composed of proliferating endothelial cells and anastomosing blood-filled channels. In most cases, a flat red spot or small mass, noted at birth or during the first six months, grows rapidly for three to six months, remains quiescent for about one year, and resolves spontaneously. ^{1–4} Resolution, which usually starts in the second year of life, is complete in about 60% of cases by age 4 years and, in up to 76%, by 7 years. ² Some superfluous skin may remain, requiring plastic surgery.

Children with infantile hemangiomas of the evelids and orbit are at risk for ocular problems, including functional amblyopia, strabismus, proptosis, and optic-nerve compression. However, the few large reported series of eyelid hemangioma have given conflicting figures on the frequency of these complications. Thus, Holland⁴ claimed that visual complications are relatively rare, whereas Robb⁵ emphasized the high incidence of amblyopia secondary to refractive errors. Also, the methods of treatment are a subject of discussion.6 Conservative management (that is, waiting for spontaneous resolution) may lead to irreversible amblyopia; several authors have advocated an active approach when this complication appears likely,⁵⁻⁹ but a schedule of management has not been established.

In a study of children with hemangioma of the eyelid, we sought to determine the frequency of complications and any relationships with the tumor's size, position, and duration, and to derive guidelines for therapy.

SUBJECTS AND METHODS

We examined the records of 125 children who in the past 20 years had attended this hospital for treatment or observation of eyelid hemangiomas. Those with flat port wine type hemangiomas were excluded, and some were unable to attend because of distance from the hospital. Fifty-one patients were studied and their parents were interviewed.

We defined amblyopia as visual acuity of 6/12 (20/40) or less, severe if 6/60 (20/200) or less, and moderate if 6/30 (20/100) to 6/12 (20/40).

We considered anisometropia of 2 diopters or more significant enough to produce amblyopia, even though some patients tolerate a higher degree. ¹⁰ As astigmatic errors may produce a specific type of amblyopia (meridional), ¹¹ in cases of astigmatism we calculated the anisometropia from the refraction in the meridian of the highest refractive error and not the spherical equivalent.

RESULTS

Twenty-seven of the 51 patients had ophthalmic complications. Amblyopia was the most common complication, present in 22, and strabismus was the next most common (Table 1). Fifteen children

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TABLE 1
OPHTHALMIC SEQUELAE IN 51 CHILDREN WITH
INFANTILE HEMANGIOMA

Complication		Percentage of Cases
Amblyopia		
Stimulus-deprivation	15	
Anisometropic or strabismic	7	44
Strabismus	17	34
Proptosis	7	14
Optic-nerve atrophy	1	2

had severe amblyopia and seven had moderate amblyopia (Fig. 1).

The study group consisted mainly of girls (75%), especially among the severely amblyopic (Fig. 1).

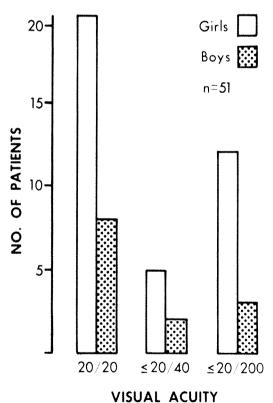


Fig. 1 (Stigmar and associates). Visual acuity in relation to sex in the study group.

Fifteen had had complete eyelid occlusion, for from one month to three years. The onset of complete closure was evenly distributed during the first year of life and averaged four months. Comparison of visual acuity with duration of eyelid closure revealed a clear correlation: the longer the occlusion, the more profound the amblyopia (Fig. 2). In 11 children, closure for three months or more had resulted in severe amblyopia in all; of four others who had complete closure for only one month, one had had moderate and three had severe amblyopia.

Eleven of these 15 children had had treatment of their amblyopia. In most

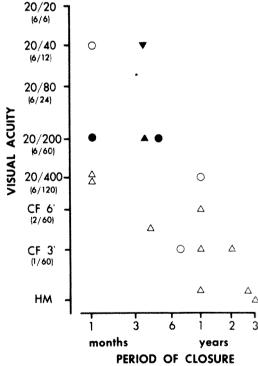


Fig. 2 (Stigmar and associates). Comparison of visual acuity with duration of complete eyelid closure (open circles) and presence of strabismus with complete eyelid closure (open triangles) in 15 patients. Solid symbols indicate values obtained in infants too young to cooperate fully. In one infant, vision improved with treatment (solid bottom triangle, initially; solid top triangle, after treatment).

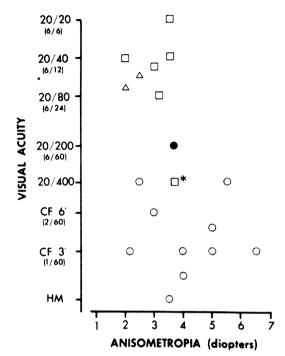


Fig. 3 (Stigmar and associates). Visual acuity in relation to degree of anisometropia. The solid circle indicates a value obtained in a very young infant. In cases with no history of stimulus-deprivation, open squares indicate anisometropia only; open triangles, anisometropia and strabismus. In cases with history of stimulation-deprivation, circles indicate complete eyelid closure; open square with asterisk, prolonged partial eyelid closure.

cases, patching of the normal eye was started at a relatively advanced age and could be tolerated only briefly. The only child for whom treatment was started early, at 4 months of age, was the only one in whom vision improved (Fig. 2).

There was a high incidence of strabismus among the 15 amblyopic children who had had complete eyelid closure (Fig. 2).

Nineteen patients had anisometropia, ranging from 2 to 6.5 diopters. There was no correlation between visual acuity and the degree of anisometropia in the 12 children in whom the amblyopia was severe, 11 of whom had experienced prolonged visual stimulus-deprivation (Fig. 3). Only one child with visual acuity of 6/120 (20/400) had not experienced prolonged complete eyelid closure, and this patient had had partial closure for two years starting at the age of 4 months. All seven children with moderate amblyopia had anisometropia of only 2 to 3.5 diopters.

Two children had simple myopia of the affected eye and six had simple hypermetropia. Eleven had mixed astigmatism (compound myopia in ten and compound hypermetropia in only one); in ten, the axis of the angle of astigmatism corresponded to the location of the tumor. The keratometer readings correlated with the total astigmatic error as determined by retinoscopy.

One third (17) of the patients had strabismus: seven had esotropia, four an exotropia, two had a vertical type of strabismus, and four had a combination of vertical and horizontal strabismus. Most of the patients had severe amblyopia, and only four had visual acuity better than 6/12 (20/40) (Table 2).

TABLE 2
FREQUENCY OF STRABISMUS IN RELATION TO VISUAL ACUITY

	Str	abismus	No	
Visual Acuity	Paralytic	Nonparalytic	Strabismus	Total
≤6/60 (20/200)	5	5	5	15
>6/60 (20/200) $\le 6/12 (20/40)$	1	2	4	7
<6/12 (20/40)	0	4	25	29
Totals	6	11	34	51

The strabismus was paralytic in six cases. Four of these patients had weakness of abduction of the affected eve; this persisted after most of the tumor had regressed, apparently because of weakness of the external rectus muscle. Three of these four patients now have normal ocular movements; in the fourth, the tumor has not fully regressed. The fifth patient had a large unilateral hemangioma involving both upper and lower evelids and could only weakly elevate that eve. This limitation persisted until long after regression of the tumor, but ocular movements eventually returned to normal. In the sixth patient, a large hemangioma in the lower evelid and orbit pushed the eve upward and restricted downward movement: the tumor has regressed but the weakness of downward rotation has persisted.

Thus, it seems that a hemangioma can affect muscle function directly by involving the muscle. However, in three of our patients who developed weakness of the external rectus, the hemangioma did not affect the region of this muscle.

The other patients with strabismus had no apparent extraocular weakness.

In seven cases the affected eye was proptosed 2 mm or more; Hertel reading averaged 4.4 mm, in the range of 2 to 8 mm. The proptosis was accompanied by strabismus in six cases and by amblyopia in five. One child had a visual field defect and slight pallor of the optic nerve suggestive of mild optic atrophy.

The predominance of females with infantile hemangiomas in general,^{2,3} and those affecting only the eyelids⁴ is reflected in our series, in the ratio of 4:1 (Fig. 1). This bias is still unexplained.

CASE REPORTS

Case 1—This patient is now 12 years old. She had a typical bulky hemangioma of the left lower eyelid, first noticed when she was 1 week old. It grew

rapidly, covering the pupil for seven months (3 to 9 months of age), then regressed slightly to incomplete coverage. The entire tumor, always well localized, was excised when she was 18 months old, shortly after the photograph reproduced here (Fig. 4, top) was taken; esotropia was noted and amblyopia treatment was started. The strabismus was corrected at age 4 years. Follow-up examination revealed a good cosmetic appearance of the eyelid. Visual acuity was R.E.: 6/6 (20/20), with glasses, and L.E.: finger counting at 3 feet, with no improvement with glasses. Refraction was R.E.: -1.25 sphere, and L.E.: -5.00 sphere, with -1.25 cylinder \times 5 degrees (Fig. 4, bottom). The optic axes were approximately straight, but there was unstable fixation of the left eye. The fundi were normal.

Case 2-This girl is now 41/2 years old. A hemangioma of the left upper eyelid started to grow when she was 6 weeks old; it never closed the eyelid fissure completely and started to resolve at age 2 years (Fig. 5). At our examination the hemangioma measured 15 × 10 mm and there was slight drooping of the eyelid. Visual acuity was R.E.: 6/6 (20/20) without glasses, and L.E.: 6/12 (20/40) without glasses and no improvement with glasses. Results of keratometry were R.E.: 44.75×90 degrees, 44.25×90 0 degrees, and L.E.: 40.5×80 degrees, 39.75×170 degrees. Refraction was R.E.: +1.00 sphere, and L.E.: +4.50 - 1.00 cylinder \times 170 degrees. There was no strabismus and she had binocular vision without stereopsis. Treatment was started with glasses and patching.

Case 3-This 5-year-old boy had had a small

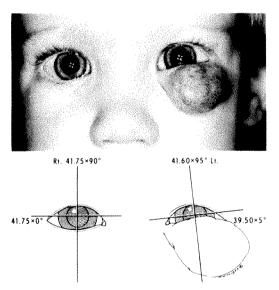


Fig. 4 (Stigmar and associates). Case 1. Infantile hemangioma, first noted at age 1 week. Top, The patient was 1½ years old. Bottom, Correlation between the tumor's asymmetric location and the angle of astigmatism.

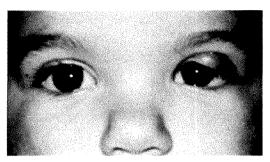


Fig. 5 (Stigmar and associates). Case 2. Patient at 2 years, just before the lesion began to resolve.

hemangioma of the right lower eyelid at birth. The diffusely growing tumor never closed the eye or pressed on the globe and it started to resolve by 2 years. The hemangioma resolved completely and the child now has normal visual functions. His visual acuity is 6/6 (20/20) in both eyes; refraction is R.E.: +2.00 -2.00 cylinder \times 0 degrees, and L.E.: +1.00-1.00 cylinder $\times 0$ degrees.

Case 4—This girl is now 15 years old. The tumor of her left upper eyelid started to grow when she was 6 weeks old. At 5 months, because closure of the eyelid fissure was imminent, the tumor was excised. By 1 year the cosmetic result was good, and she now has normal vision and normal eyelids.

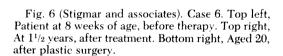
Case 5—This child, who is now 3 years old, had a diffuse hemangioma of the upper eyelid. She has severe amblyopia of her right eye, with eccentric fixation and esotropia but no anisometropia. This child had been followed-up, but too infrequently for detection of developing occlusion which occurred at 7 months and was complete for seven months. When the tumor regressed, patching was unsuccessful: untreatable amblyopia caused by stimulusdeprivation had developed.

Case 6-Our oldest patient had had a huge hemangioma as a baby 20 years previously (Fig. 6, top left). Treatment, started at 2 months, comprised the insertion of radon seeds, a sclerosant injection, and surgical excision (Fig. 6, top right). Her visual acuity is limited to counting fingers at 1 m, and despite plastic surgery, the cosmetic appearance is far from good (Fig. 6, bottom righ:).

Case 7—This boy, the youngest patient in our









series, is now almost 1 year old. His right cheek was slightly swollen at birth. At I month the hemangioma started to grow, and within four weeks it had completely closed the right eye. The patient was given prednisolone. Unfortunately, the hemangioma responded slowly, severe thrombocytopenia (Kassabach Merritt syndrome) developed, and respiratory obstruction was imminent because of pressure of the tumor, which had extended to the neck. We therefore used x-irradiation (750 rads) in addition to the prednisolone. The response was dramatic. His right eye, which had been closed for three months, began to open, and the thrombocyte count increased. The right eye was esotropic, but patching of the left eye for one month led to the development of alternating strabismus. Now, the squint has been corrected surgically, visual functions will probably be excellent, and the cosmetic result is good.

DISCUSSION

Any one or a combination of the following three factors could have been responsible for the amblyopia in Case 1: anisometropia, strabismus, and stimulusdeprivation. The most likely factor is the eyelid closure for seven months, since even brief stimulus-deprivation during the first year of life may be disastrous for visual development. The close correspondence between the degree of amblyopia and the duration of occlusion (Fig. 2), as well as the lack of correlation between the degree of amblyopia and the amount of anisometropia (Fig. 3), favor stimulusdeprivation as the main cause of severe amblyopia in Case 1.

This reasoning applies in all but one of the 15 patients with amblyopia, most of whom also had anisometropia or strabismus, or both. In most, the response to treatment was poor. Regardless of whether the cause of the amblyopia was stimulus-deprivation, strabismus, or anisometropia, or a combination of all three factors, we agree with Robb⁵ that good vision should result if therapy is initiated at an early age. An excellent result was obtained in Case 2, in whom patching was started at 4 months of age.

Anisometropia was the cause of amblyopia in Case 2 and was the major cause in others with moderate amblyopia. This case illustrates that anisometropia leads to amblyopia. Anisometropia was present in a significant number of our patients with the greater amount of hyperopia (or lesser amount of myopia) occurring in the affected eye.

All kinds of refractive errors of the affected eve produced the anisometropia. Our findings (Cases 1 and 2) concur with those of Robb,5 who first reported permanent refractive errors caused by pressure of the tumor, and close correlation between the tumor's location and the astigmatic axis, after hemangioma of the evelid in infancy. He found that hemangiomas exert pressure on the eye in a direction perpendicular to the axis of the astigmatism. Thus, anisometropia should be considered a major cause in cases of moderate amblyopia, as well as an important prognostic sign in severe amblyopia. If anisometropia exceeds 3.5 diopters, it usually predicts poor vision (Fig. 3).

As expected, strabismus was present in many of the children with the most profound amblyopia and in some of those with moderate amblyopia. The strabismus was paralytic in some, suggesting involvement of a muscle or its nerve. In comparison with stimulus-deprivation and anisometropia, strabismus plays a minor role in the production of amblyopia.

If one includes clinical signs such as proptosis, paralytic strabismus, blepharoptosis, and optic atrophy, 20% of our patients had persistent orbital manifestations of hemangioma involvement. Mild or moderate protrusion of the eye was the most common sign. The proptosis usually was associated with severe impairment of vision, in most cases deprivation-type amblyopia, and in one case was associated with mild optic-nerve compression and optic atrophy. This latter child had a large hemangioma of the left side of the forehead and the left eyelid that closed

the eye; there was a dense nasal-field defect, with temporal pallor of the disk. Skull radiographs revealed enlargement of the orbit, and a computed axial-tomography showed medial displacement of the optic nerve.

Proptosis is difficult to recognize in its early stages in infants.

Correlation of the extent and location of the tumor with final visual acuity yielded the following conclusions:

- 1. Any hemangioma that involves the upper or lower eyelid and leads to partial closure of the eye in infancy may interfere with vision.
- 2. The hemangiomas least likely to affect vision involve the lower eyelid, occupy only one third of the eyelid margin, and tend to resolve early.
- 3. Hemangiomas that lead to deprivation amblyopia, with or without anisometropia, occupy more than half the margin of one eyelid, tend to resolve late, and obstruct the visual axis.
- 4. The risk of isolated anisometropic amblyopia is greatest in children with local but bulky hemangiomas that are restricted to the upper eyelid, close the eye only partly, and tend to resolve late.

In Case 3, all of our prognostic criteria were favorable: there was no significant anisometropia or strabismus, the tumor was on the lower eyelid, and it occupied less than one third of the eyelid margin. Thus, this presents only a cosmetic problem and should be managed as an infantile hemangioma at any other site. If resolution is incomplete, cosmetic surgery should be undertaken after the age of 8 years.

Case 4 supports our contention that, if the tumor is local and may affect vision (by occlusion or anisometropia), it should be excised. We believe that in some of our patients (Case 2), treatment with glasses and patching probably could have been obviated by early surgery.

Case 6 illustrates an older type of treat-

ment. According to newer principles (Case 7), the patient would have begun systemic corticosteroid therapy and local treatment of the tumor would be instituted at an early stage of the tumor's development.⁷

In Case 7, because the extent of hemangiomatous involvement ruled out surgery, corticosteroid treatment seemed ineffectual, and the child's general condition was serious, we had no option but to try irradiation in relatively high dosage. The risk of severe side- and after-effects with this therapy had to be weighed against the possibility of profound unilateral amblyopia and even, at one stage, the child's survival.

The most important conclusion to emerge from this study is that each child should be treated individually. Additionally, patients with hemangioma involving an eyelid should have frequent check-ups during the first year of life. If the tumor does not interfere with vision, leave it alone. If the tumor is localized, obscures vision, or has produced significant anisometropia, excise it. If the tumor is diffuse and evelid closure is imminent, further treatment such as corticosteroids or xirradiation must be considered. More efficacious, safe treatment to cause regression of these tumors is needed. Slings may be required temporarily to raise the evelid and prevent amblyopia. Correct anisometropia and treat amblyopia as soon as possible. Delay cosmetic repair until after 8 years of age.

SUMMARY

The major findings in a study of 51 infants and children with infantile hemangioma of the eyelid were as follows:

Visual complications occurred in 27 patients.

The most common complications were amblyopia (in 22) and strabismus (in 17).

Amblyopia of 6/60 (20/200) or less was probably caused by stimulus-deprivation, but amblyopia in the range of 6/30 (20/100) to 6/12 (20/40) was likely caused by anisometropia or strabismus.

Eyelid occlusion of six months or more invariably resulted in amblyopia of 6/60 (20/200) or less. Occlusion for even one month carried a risk of amblyopia.

Each child must be considered individually for therapy, which must be started as early as possible. Patients should receive careful follow-up from the beginning to prevent severe amblyopia. For difficult cases, we need more efficacious, safe methods to induce regression of these tumors.

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ECTROPION OF THE LOWER EYELID SECONDARY TO MÜLLER'S MUSCLE-CAPSULOPALPEBRAL FASCIA DETACHMENT

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An ectropion of the lower eyelid usually occurs as a result of horizontal laxity of the eyelid, cicatricial changes of the skin, or paralysis of the orbicularis muscle. The purpose of this report is to present another cause of ectropion, detachment and recession of Müller's muscle and capsulopalpebral fascia.

CASE REPORT

In September 1976, a 75-year-old woman was examined by me for visual problems created by complete blepharoptosis and lateral canthal nasal displacement bilaterally (Fig. 1, top left). The patient was uncertain whether these deformities had existed all her life or occurred in recent years. The palpebral fissure width in the primary position of gaze was 3 mm on each side with fixation of the frontalis muscle; levator function, measured by the excursion of the eyelid from downgaze to upgaze, was 10 mm in both eyes. The eyelids did not elevate when 10% phenylephrine was instilled into the cul-de-sacs, thereby eliminating the possibility of doing a successful Müller's muscle-conjunctival resection blepharoptosis procedure.1 The upper eyelids were thin and creases were approximately 20 mm above the eyelid margins, suggesting a levator aponeurosis and Müller's muscle detachment and recession.2

The upper eyelid creases are created by attachments from the levator aponeurosis to orbicularis and skin and as the levator aponeurosis recesses into the orbit, it drags the crease with it and places it more superiorly. When Müller's muscle and levator aponeurosis detach from the tarsus and recess within the orbit, the eyelid becomes very thin, as it only consists of orbicularis muscle, skin, and conjunctiva above the tarsus; frequently, iris markings can be seen through the eyelid.

In November 1976, both upper eyelids were explored, and I found Müller's muscle and levator aponeurosis detached from tarsus and recessed into

the orbit 18 mm above the superior tarsal borders. I reunited these tissues to the tarsus, and eyelid creases were created. During the same surgical sitting, the lateral canthi were sutured to the lateral canthal tendons near the lateral orbital rims. Postoperatively, the upper eyelids and lateral canthi were in satisfactory positions, but a severe left lower eyelid ectropion occurred (Fig. 1, top right).

In Lanuary, 1977, L. surgically, explored the left

In January 1977, I surgically explored the left lower evelid (Fig. 2A). A 4-0 black silk suture passed through skin, orbicularis muscle, and superficial tarsus beneath the central lower eyelid lashes pulled the evelid upward during the procedure (Fig. 2B). The skin was incised with a No. 15 Bard-Parker blade across the lower evelid, approximately 6 mm below the eyelid margin (Fig. 2B). The skin was pulled forward with forceps and the orbicularis muscle was buttonholed centrally and then severed nasally and temporally along the skin incision site (Fig. 2C). By pulling the central skin forward with forceps, the orbicularis muscle can be safely severed without injury to any of the other eyelid structures. Orbicularis muscle that is tightly adherent to skin comes forward when the skin is pulled anteriorly; meanwhile, the more internal eyelid tissues such as septum, capsulopalpebral fascia, Müller's muscle, and conjunctiva remain posteriorly and inward as they surround and follow the globe. The inferior skin-orbicularis muscle flap was pulled downward and outward, while the traction suture pulled the evelid margin upward. Only thin, redundant conjunctiva existed for at least 15 mm below the inferior tarsal border all across the eyelid. Blunt dissection with a cotton-tipped applicator applied under the orbicularis muscle and aimed toward the inferior orbital rim facilitated isolation of the orbital septum about 8 mm below the inferior tarsal border (Figs. 2D and E). The orbital septum was tagged with a 4-0 black silk suture and was verified as septum by pulling it upward and feeling a firm resistance on palpitating its attachment to the inferior orbital rim. Blunt dissection with a cotton-tipped applicator applied against and inferior to the septum aided in the identification of the recessed end of Müller's muscle and capsulopalpebral fascia, which were isolated 15 mm below the inferior tarsal border (Fig. 2F).

The recessed edges of Müller's muscle-capsulopalpebral fascia were then attached to inferior tarsus with three 6-0 black silk mattress sutures (Fig. 2G), and the skin was closed with a continuous 6-0 black silk suture (Fig. 2H).

The ectropion was completely corrected by this procedure and the eyelid has remained normal for eight months postoperatively (Fig. 1, bottom right).

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Fig. 1 (Putterman). Top left, Preoperative photograph of patient with bilateral upper eyelid blepharoptosis and lateral canthal nasal displacement. (Frontalis muscle is not fixated). Top right, Severe left lower eyelid ectropion after bilateral levator aponeurosis-Müller's muscle advancement-and-tuck blepharoptosis procedures and after lateral canthaltendon tucks. Bottom right, Relief of ectropion after attaching recessed Müller's muscle and capsulopalpebral fascia to tarsus.





DISCUSSION

Detachment of the levator aponeurosis from the tarsus has been described as a cause of acquired blepharoptosis.2 Detachment of the capsulopalpebral fascia (the analogue of the levator aponeurosis in the lower eyelid) has been shown to lead to entropion.3 I have theorized that the entropion is caused by the detached, recessed capsulopalpebral fascia inserting and acting on Müller's muscle and conjunctiva about 12 mm below the tarsus, rather than at its normal insertion on the anterior tarsus. Additionally, I have noted in 18 patients another cause of acquired blepharoptosis—detachment of both Müller's muscle and levator aponeurosis from the tarsus and recession of these tissues 15 to 20 mm above the superior tarsal border. The reattachment of Müller's muscle and levator aponeurosis to tarsus corrected the blepharoptosis in these patients.

In the present case, a severe left lower eyelid ectropion occurred after successfully relieving bilateral blepharoptosis and lateral canthal avulsions by reattaching the recessed Müller's muscle and levator aponeurosis to tarsus and the nasally displaced lateral canthi to the lateral orbital rims. It was difficult to understand why an ectropion occurred after tightening the eyelid in a horizontal direction, for this procedure usually corrects senile acquired ectropion. Also, I doubted that horizontally shortening this eyelid with a routine senile acquired ectropion procedure would correct the ectropion, as the eyelid was already horizontally tight. On analyzing the possible cause of this ectropion, I deduced that the only reason could be detachment of Müller's muscle and capsulopalpebral fascia from the tarsus secondary to edema resulting from the blepharoptosis and lateral canthal surgery. I theorized that only redundant con-

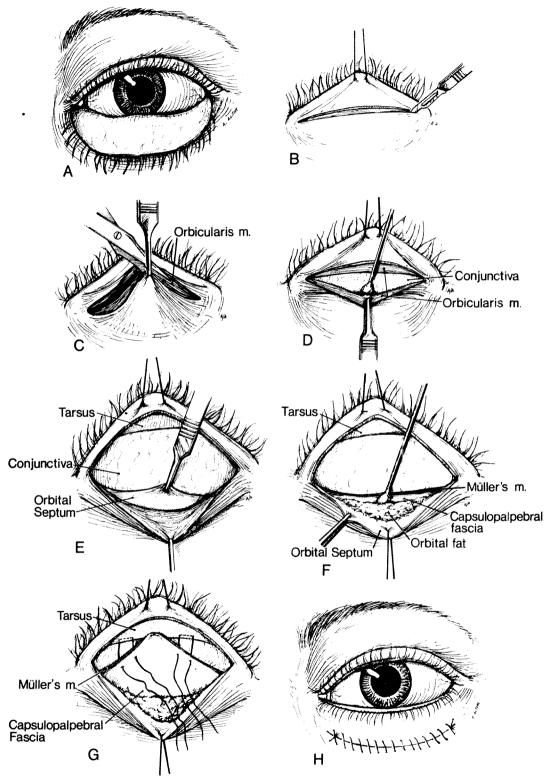


Fig. 2 (Putterman). A, Ectropion in described case. B, Skin incision at level of inferior tarsal border. C, Severing orbicularis along skin incision site. D, Blunt dissection of orbicularis muscle from underlying tissue by applying cotton-tipped applicator under orbicularis muscle, aimed toward inferior orbital rim. E, Isolation of orbital septum. F, Isolation of capsulopalpebral fascia and Müller's muscle 15 mm below inferior tarsal border. Only redundant conjunctiva existed between recessed Müller's muscle and capsulopalpebral fascia and inferior tarsal border. G, Attachment of recessed Müller's muscle and capsulopalpebral fascia to tarsus. H, Skin closure.

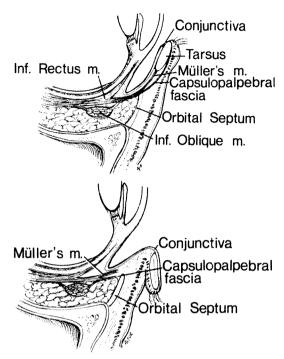


Fig. 3 (Putterman). Top, Cross section of normal anatomy of lower eyelid with insertion of Müller's muscle on inferior tarsal border and of capsulopal-pebral fascia on anterior superior surface of tarsus. Bottom, Cross section demonstrating ectropion in described case secondary to detachment of Müller's muscle and capsulopalpebral fascia.

junctiva was attached to the tarsus, which could not pull it downward; therefore, the inferior tarsal border rotated upward and outward (Fig. 3, top and bottom).⁴ This theory was proved by surgically exploring the lower eyelid and finding that Müller's muscle and capsulopalpebral fascia were detached from the tarsus and recessed 15 mm below the inferior tarsal border. The theory was further supported in correcting the ectropion by reattaching Müller's muscle and capsulopalpebral fascia to the inferior tarsus.

It is possible that edema and degeneration of eyelid tissues were the original causes of the bilateral blepharoptosis and lateral canthal nasal displacement; the patient also may have had a predisposition to detachment of Müller's muscle and capsulopalpebral fascia.

SUMMARY

A patient developed severe lower eyelid ectropion after a bilateral levator aponeurosis and Müller's muscle advancementand-tuck blepharoptosis procedure and bilateral attachment of the lateral canthi to the lateral canthal tendons. The cause of this ectropion was detachment of Müller's muscle and capsulopalpebral fascia from the inferior tarsus and recession of these tissues into the orbit. This left the inferior tarsal border with only redundant conjunctiva attached to it, which could not maintain it in a downward direction; thus, an ectropion occurred.

Müller's muscle and capsulopalpebral fascia were detached from the inferior tarsus and recessed 15 mm into the orbit. Reattaching Müller's muscle and capsulopalpebral fascia to the inferior tarsus relieved the ectropion.

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INTRALESIONAL CORTICOSTEROID THERAPY OF CHALAZIA

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Chronic inflammations of the meibomian glands (chalazia) are cosmetically disfiguring lesions that may recur and that frequently fail to respond to hot soaks and topically administered antibiotics. Although minor, chalazion surgery is inconvenient and frightening to many patients. The proven effectiveness of intralesional corticosteroid treatment in a variety of acute and chronic inflammatory skin lesions prompted us to examine its value in the treatment of 12 patients with 17 chalazia.

SUBJECTS AND METHODS

We studied 12 adult patients who had come to the outpatient clinic for the treatment of a mass lesion of one or more eyelids. The clinical diagnosis of chalazion was based on clinical history and the appearance and location of the lesion. Most patients had experienced rapid onset of a painful inflamed mass that had reached a stationary size. We informed the patients of the nature of the treatment and obtained their consent.

Ten milligrams per milliliter of triamcinolone acetonide was diluted with normal saline to a concentration of 5 mg/ml for intralesional use. The suspension was drawn up into a 1-ml tuberculin syringe fitted with a 27- or 30-gauge 5/8-inch nee-

dle. The injection was administered without local anesthesia. The needle was placed in the center of the chalazion, which was approached either from the skin or conjunctival surface. In the latter case, a chalazion clamp gently placed around the mass grasped the full thickness of the eyelid, which was then everted. A volume of 0.05 to 0.2 ml of the corticosteroid suspension was injected. The clamp, if used, was then removed. Topical antibiotic coverage with sulfisoxazole diolamine drops was discontinued after three days.

Patients were re-examined at two days, one week, two weeks, and four weeks after the injection. If the lesion had not decreased significantly in size on subsequent visits, a second injection was administered. Photographs of the lesions were taken at each visit.

RESULTS

We treated seven women and five men. Their mean age was 36 years, with a range of 20 to 54 years. Two patients had a past history of chalazia. The right eyelids were involved in six cases: four lesions were located in the upper eyelid and two in the lower. Four patients had involvement of the left lower eyelid. Two patients had bilateral lesions. Lesions measured from 2 to 10 mm in diameter. Eight patients had one chalazion, three had two, and one had a total of five involving all eyelids. In all cases, the lesion had lasted from six weeks to one year, with a mean duration of 6.1 months. A total of 17 chalazia were injected with a suspension of 5 mg/ml of triamcinolone acetonide according to the method described above (Fig. 1). In two patients

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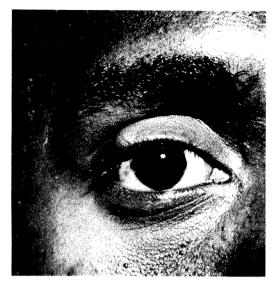




Fig. 1 (Jakobiec and associates). Left, Thirty-four-year-old man with a mass in the right upper eyelid for five months. Right, Four weeks later after two injections of intralesional corticosteroids, lesion has completely resolved.

who had two chalazia, one chalazion was injected with corticosteroid and the second served as a control and received only hot soaks and sulfisoxazole diolamine drops. The corticosteroid-treated lesions resolved with no change in the conservatively treated lesions (Fig. 2).

Nine lesions received a single injection; of these, seven resolved without further treatment with corticosteroid. Resolution was inferred by disappearance of a palpable mass or a reduction in size of the mass to 1 mm or less in diameter. Resolution occurred rapidly, generally one to





Fig. 2 (Jakobiec and associates). Left, Twenty-three-year-old woman with two chalazia in the right upper eyelid and one in the left upper eyelid. Right, Both lesions in the right upper eyelid resolved after only one injection, but the lesion in the left upper eyelid persisted under conventional therapy during the four weeks of follow-up.

two weeks after injection. Two chalazia showed resolution within two weeks of a single injection; however, one to two weeks later the lesion had either recurred or'a new one had developed adjacent to it. Eight lesions were reinjected, one at two days following initial treatment, six at one week, and one at one month. Six of these eight lesions resolved within two weeks after reinjection. Two chalazia failed to respond to two injections of corticosteroids. All lesions that improved have remained so for six weeks, with the exception of the two cases of recrudescence. The only complication in the series occurred in a black man who developed a yellow eyelid deposit at the site of transcutaneous injection.

DISCUSSION

Dermatologists routinely and successfully use the intralesional injection of corticosteroids for the treatment of a wide variety of acute and chronic inflammatory conditions of the skin including acne cysts, plagues of psoriasis and lichen planus, granulomas of sarcoidosis, and nodules of chronic arthropod bite reactions.1-3 The ophthalmic community has accepted the periocular injection of corticosteroids primarily for ocular inflammatory disorders such as uveitis, episcleritis, or chorioretinitis.4 Complications have been few, some of the most serious being prolonged increase of the intraocular pressure,⁵ unsightly yellow discoloration of the epibulbar tissue, 5,6 and atrophy of the orbital fat.7

Leinfelder⁸ first proposed treating chalazia with corticosteroid injections. However, he treated acute and subacute chalazia only to convert them into more compact and easily resectable nodules. Rather than directly injecting lesions, he introduced 0.25 ml of methylprednisolone acetate subconjunctivally in the quadrant containing the chalazion and

reported that the lesions decreased in size.

Chalazia are predominantly composed of corticosteroid-sensitive histiocytes, multinucleated giant cells, lymphocytes, plasma cells, polymorphonuclear leukocytes, and eosinophils. The local injection of corticosteroids has the desirable effects of suppressing additional inflammatory cells and impeding chronic fibrosis and scar formation, which typically appears as a small, firm, nontender nodule after the resolution of the acute chalazion.

Our results suggest that intralesional injection of corticosteroids into chalazia is an effective, rapid form of treatment without significant complications. Of 17 lesions that were injected, 13 displayed prompt and lasting resolution within one to two weeks after one or two injections. Two lesions did not subside at all. Another two lesions subsided after injection, but either they recurred or a second neighboring lesion occurred. The only complication was in a black man who developed a vellow deposit at the site of injection. This blemish has been slowly resolving and may be avoided in the future by transconjunctival injections. Atrophy of skin secondary to local administration of corticosteroid is a recognized complication in the treatment of cutaneous lesions, but was not observed after treatment of chalazia. It is almost always temporary, and the appearance of the skin returns to normal within several months. Crystalline or insoluble preparations of corticosteroid (so-called slow absorbing "depot" forms) may produce permanent atrophic changes and therefore are not recommended for the treatment of chala-

The use of the chalazion clamp with eyelid eversion and the injection itself caused little discomfort to the patients. The patients who had previously undergone surgery for excision of chalazia found injection a preferable mode of treatment. The speed and simplicity of the procedure are intrinsically appealing features because the cumbersome use of hot compresses and even eventual surgery were circumvented. Patching of the eye is unnecessary, and the entire procedure takes less than five minutes. This form of treatment is particularly suitable for chalazia located near the lacrimal drainage apparatus because surgery in this area may lead to serious complications.

The pain the patient feels on injection of the lesion is no greater than on xylocaine injection before a surgical procedure. We recommend initial injections of 0.05 to 0.2 ml of 5 mg/ml of triamcinolone acetonide. A second injection two days later will facilitate complete resolution of the lesion because partial response after the first injection permits the introduction of a larger volume of corticosteroid. If the patient is unable to return in two days, he may do so a week later, when the need for re-injection can be assessed in the light of the response to the first treatment. Nine of the 13 successfully treated lesions in our series responded to only one injection.

We used a soluble aqueous preparation rather than an insoluble, crystalline suspension of corticosteroid to minimize complications of hypopigmentation and atrophy of the treated skin. A transconjunctival injection provides a further safeguard against these complications. Depending on the size and location of the lesion, both transconjunctival and transcutaneous approaches might be combined in selected patients.

We do not believe that the fear of injecting a meibomian gland carcinoma is a realistic reason for avoiding intralesional corticosteroid therapy for chalazia. Most chalazia occur in younger patients, are painful, and enlarge in a short time. Conversely, meibomian gland carcinomas grow insidiously, but steadily, and tend

to occur in older patients.⁹ The dramatic short-term response we obtained from the intralesional injection of chalazia would not be expected in a malignant tumor. Any friable tissue obtained during surgery on a chalazion that has failed to respond to corticosteroid injections should, of course, be examined histopathologically.

SUMMARY

Twelve adult patients with 17 chalazia underwent trial intralesional injection of triamcinolone acetonide. Seven chalazia resolved within two weeks after only one injection, and another six after two injections. Two lesions failed to respond to two injections, and two lesions responded to one injection, but either recurred or another lesion developed. Patients were satisfied with the procedure, which appears to be a safe, convenient, and effective alternative to chalazion surgery.

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GENERALIZED FIBROSIS OF THE EXTRAOCULAR MUSCLES

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One of the earliest descriptions of the combination of congenital blepharoptosis and markedly restricted eye movements is attributed to Baumgarten in 1840. However, the classical report by Heuck¹ in 1879 described a familial occurrence of congenital bilateral blepharoptosis and absence of extrinsic ocular muscle function and included a postmortem examination of one of the affected family members. Heuck described a posterior membranous insertion of all the rectus muscles, with an anterior and medial insertion of the superior and inferior oblique muscles.

Brown² coined the term "general fibrosis syndrome" in 1950 after evaluation and treatment of three sporadic cases with clinical features similar to those described by Heuck and others.^{3–8} Although the disorder is rare, numerous reports have been published since 1840 with descriptions of some cases occurring in families, others sporadically.

The confusion regarding generalized fibrosis of the extraocular muscles is both the product and cause of the terminology applied to it. The terms include: general fibrosis syndrome, congenital ophthalmoplegia, strabismus fixus, abiotrophic ophthalmoplegia externa, and ophthalmomyopathia congenita. Some of the names are used to describe specific disorders not associated with general fibrosis, but the confusion in terminology persists.

Laughlin⁸ characterized the syndrome as encompassing the following features: (1) fibrosis of the extraocular muscles; (2)

fibrosis of Tenon's capsule; (3) adhesions between muscles, Tenon's capsule, and globe; (4) inelasticity and fragility of the conjunctiva; (5) absence of elevation or depression of the eyes; (6) little or no horizontal movement; (7) eyes fixed 20 to 30 degrees below the horizontal; (8) blepharoptosis; (9) chin elevation; and (10) condition present at birth. Other neurologic or anatomic lesions are usually absent. The disease is almost always bilateral, although sometimes unilateral. 9

The differential diagnosis of the syndrome includes double elevator palsy, Brown's superior oblique tendon sheath syndrome, progressive external ophthalmoplegia, Duane's syndrome, and myasthenia gravis. Other diagnostic possibilities include orbital periostitis and entrapment from an orbital floor fracture. These disorders can be differentiated by history and clinical examination, including forced ductions.

Only a few histopathologic reports are available. They describe either total replacement of muscle tissue with fibrous tissue or marked degenerative changes in the muscles. 7.8.10 Electron micrographs have shown active fibrocyte proliferation and absence of striated muscle tissue. 11 Associated abnormalities, such as coloboma, mixed hyperopic astigmatism, retinitis pigmentosa, and amblyopia have sometimes been noted.

We describe herein the clinical, anatomic, and histopathologic findings and the surgical results in four patients with the general fibrosis syndrome.

CASE REPORTS

Case 1—A boy, born in July 1967 at term without complication, had bilateral blepharoptosis and strabismus. The family history was noncontributory for eye disease. The patient developed normally and, although shy, interacted normally with others.

The patient was first seen at the Jules Stein Eye

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Institute at age $2^{1/2}$ years. The levator muscle was not functioning in either upper eyelid. The eyes were positioned down and out, for which the patient compensated by assuming a chin-up head posture (Fig. 1). Attempted upgaze resulted in a slight convergence of both eyes. Atropine refraction at age 5 years was R.E.: $-0.50 + 4.0 \times 90$; L.E.: $+1.50 + 2.0 \times 90$. Spectacles with the full correction were prescribed.

When the child was 6 years old, ocular examination under general anesthesia revealed a 75-prism diopter exotropia (both eyes divergent) associated with a 50-diopter right hypotropia and a 45-diopter left hypotropia as measured by the prism reflex test (Krimsky's method). Because of the fixed globes, eye position in all four patients was measured by the Krimsky method instead of by the prism alternate cover test. On forced ductions, both globes were markedly restricted in all fields. During surgery, on exposing the right inferior rectus muscle, we found numerous adhesions between the muscle, Tenon's capsule, intermuscular septum, and globe. The right inferior rectus muscle inserted into the globe in a broad, oblique way angulated medially 7 mm from the corneoscleral limbus at its closest point. The operation performed was a 10-mm recession of the right inferior rectus muscle combined with a mar-

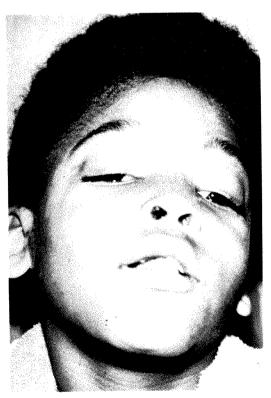


Fig. 1 (Apt and Axelrod). Case 1. Preoperative chin-up head position with blepharoptosis and exohypotropia.

ginal myotomy, and a resection of the right superior rectus muscle of 4.5 mm with advancement of 1 mm. The 10-mm recession site was reached by permitting the disinserted muscle to retract, which allowed the globe to rise to the primary position. A stay suture held the globe up postoperatively. The inelastic conjunctiva was recessed, which left the sclera bare between the original right inferior rectus insertion site and the corneoscleral limbus.

Surgery on the inferior and superior rectus muscles of the left eye when the patient was 7 years old revealed similar findings. The left superior rectus muscle was resected 4.5 mm, and the left inferior rectus muscle was disinserted and permitted to retract until the globe rose to the primary position. Stay sutures maintained the raised globe position postoperatively. The conjunctiva was closed by the bare sclera procedure.

Since the globe had become hypotropic again when the child reached 8 years of age, lysis of adhesions surrounding the right inferior rectus muscle was performed. Two months later, to correct the exotropia, the left lateral rectus muscle was recessed 10 mm, and the left medial rectus muscle was resected 6.5 mm. Both muscles were taut, wide (12 mm), and inserted obliquely from below instead of horizontally. A stay suture kept the globe adducted postoperatively; the bare sclera conjunctival closure was performed.

When the patient was 8½ years old, surgery on the horizontal muscles of the right eye revealed findings similar to those in the left eye. The right lateral rectus muscle was allowed to recede 10 mm behind its original insertion, and the right medial rectus muscle was resected 7 mm. We again used a stay suture to keep the globe adducted postoperatively and a bare sclera closure.

Three months later we performed a brow suspension blepharoptosis with a fascia lata sling on both upper eyelids.

The present eye position is excellent (10 prism diopters right hypotropia with 0 to 8 prism diopters left exotropia), and the abnormal chin-up head position is no longer evident (Fig. 2).

Case 2—A boy was born in October 1968 with bilateral blepharoptosis and strabismus. He had no other physical abnormalities. The family history was noncontributory for ocular problems. The boy developed and functioned normally, even though the eyes were fixed in the down and out position for which he used a compensatory chin-up head position (Fig. 3).

The refractive error with atropine was R.E.: +0.50 +1.00 × 90; L.E.: +1.50 sphere. The patient had 2 to 3 mm of levator muscle function in the left upper eyelid and no levator muscle function in the right upper eyelid. On rotations, only slight abduction movement of each eye could be elicited. The right eye measured 20 prism diopters of exotropia with 50 prism diopters of hypotropia; the left eye, 35 prism diopters of extropia and 60 prism diopters of hypotropia.

Examination under general anesthesia when the child was 5 years old revealed marked restriction to forced duction in all fields and greater restriction in

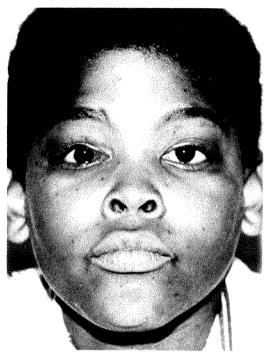


Fig. 2 (Apt and Axelrod). Case 1. Postoperative normal head position, eyelid level, and alignment of eyes.

the vertical plane. During surgery, the left inferior rectus muscle was found 8 mm from the corneoscleral limbus, and it was recessed 8 mm by letting it retract after disinsertion from the globe. The left superior rectus muscle was taut and inserted obliquely onto the globe, and it was resected 5 mm. A stay suture kept the globe up for one week after the operation. The fibrotic conjunctiva was recessed, which left bare sclera exposed.

When the patient was 5½ years old, on forced duction maneuvers, examination of the right eye under general anesthesia revealed findings similar to those encountered in the left eye. The right inferior rectus muscle was recessed 13 mm after disinsertion from the globe, and the right superior rectus muscle was resected 5 mm. As in the left eye, stay sutures and the bare sclera conjunctival closure were used.

Since hypotropia (30 prism diopters) recurred one year later, the left inferior rectus muscle was explored. Adhesions were lysed, and the muscle was re-recessed to a point 12 mm behind the original insertion.

Forced ductions of the left eye under general anesthesia when the child was 7 years old revealed marked restriction to movement in all fields. The horizontal rectus muscles were inserted broadly onto the globe obliquely from below. They were taut and appeared fibrotic. The left lateral rectus muscle was recessed 10 mm, and the left medial rectus muscle was resected 6 mm. Bare sclera conjunctival closure was used.

When the patient was 8 years old a Fasanella-Servat levator muscle resection procedure was performed on the right upper eyelid. The position of the eye and eyelid is excellent and is 0 to 3 prism diopters of exotropia on distance fixation (Fig. 4).

Case 3-A boy was born in November 1963 after an uncomplicated pregnancy and delivery. Striking bilateral blepharoptosis, hypotropia, and a compensatory chin-up position were noted at birth. When we first saw the patient here at age 9 months, we observed levator muscle function of 3 to 4 mm in both upper eyelids. The right eye was 30 prism diopters hypotropie; only a trace amount of abduction, adduction, and elevation was present. The left eye was 20 prism diopters hypotropic; it had a small amount of abduction, but almost no elevating function (Fig. 5). A fine rotary nystagmus was present on attempted voluntary eye movement. Additionally, a reverse Bell's phenomenon was noted. Atropine refraction revealed R.E.: +2.50 sphere; L.E.; +1.50 sphere. The family history was noncontributory for ocular disease. The patient's intellectual and physical abilities were normal.

Ocular examination under general anesthesia when the child was $2^{1/2}$ years old revealed a moderate restriction to forced elevation in both eyes and mild restriction to rotation in the horizontal plane. The lower fornices were foreshortened and contracted by subconjunctival fascial adhesions extending to the globe.

On surgical exploration, the right inferior rectus muscle, found inserted 10 mm behind the corneoscleral limbus, was recessed 4 mm after lysis of adhesions surrounding the muscle. The left inferior rectus muscle, inserted 8 mm behind the corneoscleral limbus, was recessed 4 mm after lysis of



Fig. 3 (Apt and Axelrod). Case 2. Preoperative chin-up head position, with blepharoptosis and exohypotropia.

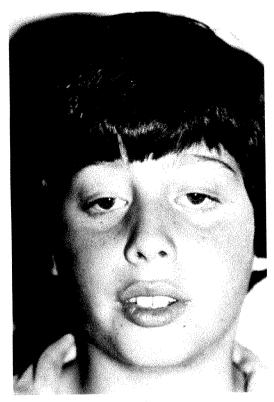


Fig. 4 (Apt and Axelrod). Case 2. Postoperative correction of abnormal head position, with improved eyelid level and alignment of eyes.

adhesions surrounding the muscle. When the patient was 8 years old, the right inferior rectus muscle was reexplored because of recurrence of hypotropia. Adhesions around the muscle were lysed, and the muscle was recessed to a point 14 mm behind the corneoscleral limbus. Additionally, a tenotomy was performed on the right inferior rectus muscle. We made a bare conjunctival closure and used a stay suture to keep the eye temporarily elevated.

Because of recurrence of hypotropia in the left eye, surgery was performed on the child at age 12½ years to lyse adhesions around the left inferior



Fig. 5 (Apt and Axelrod). Case 3. Preoperative chin-up head position with hypotropia and blepharoptosis.

rectus muscle and to re-recess the muscle to 14 mm behind the corneoscleral limbus. The conjunctiva was kept recessed with interrupted sutures, and a stay suture was used to keep the eye up postoperatively.

Although the eye position has improved greatly, further eye muscle surgery is contemplated for the residual exohypotropia (Fig. 6).

Case 4—A boy, born in November 1964 after an uncomplicated pregnancy and delivery, had an exotropia and blepharoptosis. Bilateral inguinal hernias and unilateral cryptorchism were the only other abnormal physical findings. The right upper eyelid had levator muscle function of 8 mm, and the left upper eyelid, 7 mm. The exotropia measured 50 prism diopters. The eyes could adduct only to the midline, but could abduct almost completely. On attempted lateral gaze to either side, the adducted eve depressed, the globe retracted, and the palpebral fissure narrowed. Infraduction was reduced in both eyes. The nonfixing eye was 50 prism diopters exotropic and slightly hypertropic. The patient compensated by turning his head (Fig. 7). In addition to the general fibrosis syndrome, the differential diagnosis before surgery included Duane's syndrome associated with exotropia.

On forced duction maneuvers under general anesthesia when the child was 9½ years old, the globe position measurements were the same. We encountered marked restriction to adduction of both eyes, but forced ductions in all other fields were normal. The left lateral rectus muscle appeared fibrous and was extremely tight against the globe. The muscle was wider than normal (12 mm), and the insertion curved anteriorly at its center and was recessed 10

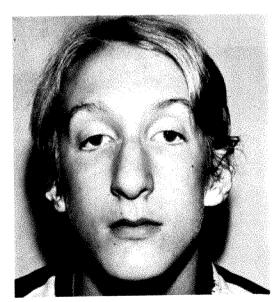


Fig. 6 (Apt and Axelrod). Case 3. Postoperative normal head position with cosmetically improved alignment of eyes. Correction of residual blepharoptosis is planned.



Fig. 7 (Apt and Axelrod). Case 4. Preoperative head turn with exotropia and mild blepharoptosis.

mm. The left medial rectus, also fibrous, was taut and had a wide insertion onto the globe and was resected $6\ \mathrm{mm}$.

When the boy was 10½ years old, surgery on the right eye revealed a 12-mm wide right lateral rectus insertion placed anteriorly, that is, 5 mm from the corneoscleral limbus at its most anterior point. The muscle was thick and fibrotic. A recession of 10 mm was performed. The right medial rectus, also fibrotic, was resected 8 mm.

Postoperatively, full adduction was evident in both eyes, although some enophthalmos still occurred during adduction. The eye position was 5 prism diopters of exotropia, and the patient's head position was normal (Fig. 8). Blepharoptosis surgery is planned, but has been delayed because of reappearance of the inguinal hernia on one side associated with an undescended testicle. Otherwise, the patient's physical and intellectual development has been normal.

MATERIAL AND METHODS

The basic surgical technique for correction of the eye position has been as follows: (1) recess the inferior rectus muscle when the eye is fixed in the hypotropic position, or recess the lateral rectus muscle

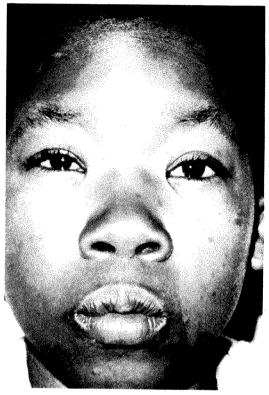


Fig. 8 (Apt and Axelrod). Case 4. Postoperative correction of strabismus and abnormal head turn.

when the eye is fixed in the exotropic position. In both instances, a sliding suture is used to reinsert the muscle on the globe as far back as necessary for the eye to assume the primary position; (2) resect the superior rectus muscle to enhance the inferior rectus muscle recession, or resect the medial rectus muscle to enhance the lateral rectus muscle recession; (3) use a traction suture to maintain the globe in its new position postoperatively; and (4) recess the conjunctiva over the recessed muscle and leave the bare sclera exposed to enhance the weakening procedure further. Blepharoptosis surgery is performed after the eyes are aligned.

In all our cases, resected tissue, including specimens of Tenon's capsule and intermuscular septum, was submitted for histopathologic study. The proximal portion of the resected muscle was tagged

to eliminate the tendonous portion from microscopic examination. The tissue was compared to a series of ten consecutive resected muscle specimens from patients with strabismus who did not have a restrictive component on forced ductions. Similarly, we evaluated only the proximal portions of the specimens.

Surgical specimens were cut for light microscopy and stained with Masson trichrome and with hematoxylin and eosin.

Before and after surgery, we identified and corrected amblyopia. Cycloplegic refractions, spectacle correction, and patching were used to obtain maximal visual function.

RESULTS

After multiple surgical procedures, the cosmetic results in the four patients were favorable (Figs. 2, 4, 6, and 8). Because we treated amblyopia in the patients initially, visual results have also been good. The most recent best-corrected visual acuity was: Case 1, 6/7.5 (20/25) in each eye; Case 2, 6/12 (20/40) in each eye; Case 3, R.E.: 6/9 (20/30), L.E.: 6/7.5 (20/25); Case 4, 6/9 (20/30-1) in each eye.

Histopathologic study of the resected tissue from the four cases with the general fibrosis syndrome revealed the presence of multiple dense bands of connective tissue. Occasionally, no muscle cells were seen in the specimens, but areas of normal striated muscle tissue were found in several cases. The muscle tissue was enveloped within a thickened and fibrotic capsule. Similarly, the intermuscular septum was thickened and fibrotic in all cases.

Muscle was present either in tight fiber bundles or in loose bundles surrounded by a delicate fibroelastic network (Fig. 9). We could not find adipose tissue infiltration or sector atrophy. The areas of muscle infiltrated by fibrous tissue were characterized by lack of orientation of fibers, which often blended imperceptibly with fibrotic Tenon's capsule.



Fig. 9 (Apt and Axelrod). Case 1. Cross section of left medial rectus muscle. Note normal-appearing fiber bundle arrangement in loose connective tissue framework. Inflammatory cells and adipose tissue infiltration are not present (Masson trichrome, X 150).

The specimens were compared with the control specimens of strabismic patients without restriction on forced ductions. Features such as variable fiber size, abundant fibroelastic tissue, multiple subsarcolemmal nuclei, and degenerative changes in the fiber reticulum were noted in both groups. We observed no inflammatory infiltrate in either group.

DISCUSSION

Our study demonstrates that satisfactory cosmesis can be obtained with appropriate extraocular muscle surgery. Amblyopia can be successfully treated when recognized early. Awkward and uncomfortable head positions can be relieved with proper alignment of the eyes. A strict sequence of surgical steps is used when the eyes are fixed in the down and out position (three of four cases), beginning with vertical, then horizontal muscle surgery, and finally, eyelid surgery. When the eyes are fixed solely in the exotropic position, only appropriate horizontal muscle surgery is done (as in Case 4). Recurrence of any appreciable degree of deviation requires reexploration, lysis of adhesions, and additional appropriate eye muscle surgery. Maximum results are facilitated with stay sutures to keep the globe in the improved position postoperatively and with bare sclera conjunctival closure.

Because the rectus muscle procedures often cause a change in relative eyelid position, blepharoptosis surgery is put off until last. Careful attempts to avoid overcorrection of the eyelids have prevented exposure problems. Repeated attempts to normalize the position of the globes and eyelids are important.

Our four patients developed normally both physically and mentally. They showed a marked improvement in demeanor after corrective eye muscle surgery.

In Case 4 strabismus involved the horizontal, rather than the vertical extrinsic eve muscles. The palpebral fissure changes and the enophthalmos on adduction suggested a Duane's syndrome associated with exotropia. However, the presence of congenital blepharoptosis and the findings of taut, abnormally inserted, fibrotic medial and lateral rectus muscles with marked restriction to adduction on forced ductions, led us to consider this case a variant of the general fibrosis syndrome. Perhaps other case reports on Duane's syndrome more accurately fall into the category of the general fibrosis syndrome.12.13 Similarly, reports of congenital inferior rectus muscle fibrosis and blepharoptosis may represent part of the spectrum of this disorder. 9 We prefer to include in the category of the general fibrosis syndrome involvement of any extraocular muscle, rather than to restrict the diagnosis to only the inferior rectus muscle.

Findings at the time of operation corroborate the discovery by Heuck¹ that the muscles in this syndrome are not only fibrous, but they insert on the globe anomalously. Light and electron microscopy reveal a marked increase in the fibroelastic component of the tissue, including Tenon's capsule and intermuscular septum, but islands of apparently nor-

mal extrinsic ocular muscle tissue can be seen.

Many researchers have attempted to explain the generalized fibrous syndrome on the basis of a nuclear, supranuclear, or myogenic defect.14-19 Unfortunately, the histologic factors used have often been based on the components of striated skeletal muscle tissue in normal and diseased states. Findings ordinarily classified as degenerative in skeletal muscle can be normal in extrinsic ocular muscle.20,21 Moreover, any theory of secondary degeneration fails to explain why the muscles have anomalous insertions. They appear to fall short of their destination. Hence, the rectus muscles are inserted posteriorly, and the superior and inferior oblique muscles are anterior and medial, as if they had experienced a maturation arrest during the differentiation of the mesodermal cone.

In the seventh week of gestation, or the 20-mm stage, the extrinsic eye muscles are just beginning to insert onto the developing sclera. They are short and round, with broad insertions. The eye is only about 1 mm long at this stage, and the muscles blend with the surrounding mesodermal tissue. Tenon's capsule develops later in association with the extrinsic ocular muscles, rather than as an independent condensation of the periocular mesoderm.

Defective maturation at or before the 20-mm stage conceivably could explain the anomalous insertions and abnormalities of Tenon's capsule found during surgery. The motor nerves already are associated with the muscles at the 20-mm stage, so that abnormal development of the nerves after this point could adversely affect muscle development. Thus, on this basis, a primary neurogenic cause cannot be excluded.

Although electromyographic studies in patients with the general fibrosis syndrome may prove worthwhile, the results may be inconsistent.²⁰ Our findings of abnormal insertion planes and extensive fibrosis of the extrinsic eye muscles and the surrounding tissue explain the inconsistencies.

The presence of islands of histologically normal muscle tissue and the absence of sector atrophy or adipose tissue infiltration disallows a myogenic cause.

The congenital appearance of the syndrome, the absence of active inflammation, and the reported occurrence in successive generations tend to exclude a postinflammatory etiology.

With a normal globe and normal intrinsic eye muscle and visual function in the absence of other neurologic dysfunction, the defects in the general fibrosis syndrome appear restricted to a faulty differentiation of the muscle cone.²³ The underlying cause remains obscure.

SUMMARY

We studied four patients with the general fibrosis syndrome. One patient had bilateral inguinal hernias and unilateral cryptorchism; the other patients had no other congenital abnormalities. The patients developed normally both neurologically and mentally. We successfully treated amblyopia and achieved good functional and cosmetic results with strabismus and blepharoptosis surgery. Histopathologic study revealed fibrous infiltration of extrinsic eye muscle and Tenon's capsule without inflammatory changes.

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EFFICACY OF BIFOCALS IN THE TREATMENT OF ACCOMMODATIVE ESOTROPIA

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When a child with esotropia and hypermetropia is given glasses based on prescription of the full cycloplegically determined refractive error, reexamination may reveal one of the following conditions: (1) orthophoria at near and distance fixation while the child is wearing glasses; (2) reduced, but not eliminated, angle of strabismus at near and distance fixation; and (3) a distance deviation which has been eliminated or nearly eliminated with glasses, while a significant deviation persists at near fixation.

This residual near deviation is thought to be caused by abnormal synkinesis between accommodation and accommodative convergence; that is, the effort to accommodate elicits an abnormally high accommodative convergence response (AC/A ratio).

Bifocals have been accepted as an effective means of reducing or eliminating this residual near deviation¹⁻⁵; however, to our knowledge, there is no valid information regarding the results of such treatment, nor are there any reported data concerning the course of accommodative esotropia treated with bifocals. Our purpose is to evaluate the effect of bifocal therapy in esotropic children.

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MATERIAL AND METHODS

This study was based on the analysis of clinical data from 84 patients who were treated with bifocals at the Department of Ophthalmology, Baylor College of Medicine, and Wilmer Institute of Ophthalmology, Johns Hopkins University Hospital and Medical School. The patients' clinical characteristics are summarized (Table 1). All patients had either orthophoria or esophoria or esotropia not exceeding 7 prism diopters at distance, and esotropia or intermittent esotropia of larger magnitude at near fixation. Before treatment was begun the refractive error was fully corrected on the basis of a recent cycloplegic refraction. Amblyopic patients were first treated with occlusion therapy; and bifocals were not prescribed until visual acuity had equalized in both eyes.

The bifocal power was titrated by adding plus lenses in +1.00 sphere steps to the patient's spectacles until the minimal power required to reduce the angle of strabismus sufficiently to permit stable fusion was determined; the patient fixated on an accommodative target at 33 cm during this procedure. The bifocal power prescribed ranged from +1.00 sphere to +3.50 sphere according to the individual's needs.

One might argue that for a measurement in an office with additional plus lenses, a patient will not relax his accommodation sufficiently to make this determination, so bifocals need to be worn for some time before their full effect on near deviation can be assessed. If this were so, further reduction of near deviation after

TABLE 1 CLINICAL CHARACTERISTICS OF 84 PATIENTS TREATED WITH BIFOCALS*

27.2 mos (1 mo - 72 mos)
17 patients
19 patients
65.4 mos (30 mos - 120 mos)
35.8 mos (3 mos - 168 mos)
R.E.: +2.9 D (+.50 to +7.5)
L.E.: +3.0 D (+.37 to +8.5)
R.E.: $+3.1 D$ (plano to $+6.8$)
L.E.: +3.2 D (plano to +8.3)

^{*}D indicates diopters.

the patient had worn bifocals for several weeks would be expected. To clarify this point, in a preliminary study we compared measurements taken during the first examination at near fixation through +3.00 lenses in 20 patients with those obtained after bifocals had been worn for 5 to 15 weeks. On the second examination, the near deviation had remained the same or had increased in all patients except two in whom the deviation had decreased. We concluded, therefore, that prescription of bifocals on the basis of measurements made during an office examination is usually justified and, in most patients, wearing bifocals for several weeks is not likely to reduce the deviation further.

In patients whose response to bifocals was doubtful, a trial with press-on lenses was often helpful before ordering the final prescription. A flat-top, executive style bifocal was prescribed to all patients, and the optician was instructed that the separation line between distance and near segments should bisect the pupil with the eyes in primary position. After the patient had obtained his glasses, the correct fitting of the bifocal was ascertained.

We estimated the stimulus AC/A with the gradient method.⁶ A change in the stimulus to accommodation was produced by measuring the angle of strabismus with the prism cover test with and without +3.00 spherical lenses while the patient, with his refractive error fully corrected, was describing the contents of a small picture, or if older, was reading the equivalent of 6/6 (20/20) letters at a viewing distance of 33 cm. The difference between these measurements represents the amount of accommodative convergence relaxed by reducing the accommodative stimulus 3 diopters; the AC/A was calculated by dividing by three the difference between the near measurement determined with and without +3.00 lenses. This two-point gradient is based on the assumption that the ratio is linear and it has been pointed out that more than two points are required to plot the AC/A accurately.6 Nevertheless, our method is useful and widely employed to estimate, for clinical purposes, whether a patient has a high or low AC/A.^{4,6-10}

During the course of treatment, cycloplegic refraction was done semiannually in all patients with either 1% atropine sulfate or 1% cyclopentolate hydrochloride, and the distance and near correction adjusted if the refractive error had changed. On each return visit, the near deviation was remeasured with and without minus lenses held over the bifocals. If the patient was able to maintain fusion at

TABLE 2 Long-term effect of bifocals in 84 patients

Group	Effect	No. of Patients
1	Fuses at near and distance without bifocals	12
2	Progressive improvement of fusional control, needs less bifocal power, still under treatment	19
3	Total dependence upon bifocals without improvement of fusional control	39
4	Deterioration of binocular function while	30
	treated with bifocals	14
	Total	84

near fixation with less bifocal power than he was currently wearing, the bifocal power was reduced.

RESULTS

We recorded the long-term effects of bifocals in these 84 patients (Table 2). Patients in Group 1 no longer required bifocals to maintain steady fusion at near and were considered cured. Patients in Group 2 manifested improved fusional control of their near deviation and required less bifocal power than at the beginning of therapy; they still receive treatment, and can be expected to improve further. Patients in Group 3 were fusing through their bifocal segments but a reduction of bifocal power was not possible in spite of prolonged therapy, and they were completely dependent on their bifocal lenses in maintaining fusion at near fixation. Patients in Group 4 showed an initial good response to bifocals, but they slowly lost fusional control of their near deviation while on maximal therapy (+3.50 sphere bifocals).

All clinical characteristics at the onset of therapy that would indicate which patients were likely to benefit from bifocals are listed (Table 3). Most of these data are surprisingly uniform. Between the four groups there is little, if any, variation in the age at the beginning of

therapy, the duration of therapy, the incidence of congenital esotropia and of previous surgery, the refractive error, and the angle of the deviation at near and distance fixation at the beginning of therapy. However, the AC/A ratios were highest and good results from orthoptic therapy (aimed at eliminating suppression, dissociating convergence from accommodation, and increasing the amplitude of divergence) were obtained most often in patients in Groups 1 and 2.

We analyzed the data further to identify the factors that led to cure, improvement, deterioration. Improved binocular function in patients of Groups 1 and 2 was brought about by a spontaneous decrease of the near deviation that permitted fusional control of the residual deviation or by an increase in fusional divergence amplitudes with the near deviation remaining the same. Since it is known that hypermetropia tends to increase in children of the age being studied,11,12 one may argue that a decrease of the near deviation could have been caused by an increase in the power of the distance correction during the course of therapy. However, the mean refractive error (spherical equivalent) did not significantly increase in most patients during this period, and for that reason the decrease of the near deviation could not have been

TABLE 3
CLINICAL CHARACTERISTICS IN PATIENTS GROUPED ACCORDING TO THERAPEUTIC RESULTS

	Group	Group	Group	Group
Characteristics	1	2	3	4
Age at beginning of therapy* (mos)	70	60	68	59
Incidence of congenital esotropia	3	5	7	2
Previous surgery	3	2	12	2
Duration of therapy* (mos)	58	34	45	40
Refractive error*				
R.E.	+3.00 D	+2.50 D	+3.00 D	+3.00 D
L.E.	+3.00 D	+2.50 D	+3.00 D	+3.00 D
AC/A*	7.2:1	7.3:1	6.1:1	4.8:1
Esodeviation at onset of therapy				
through distance correction*				
Near	26	30	25	19
Distance	6	7	6	7
Esodeviation at end of therapy*				
Near	11	21	25	27
Distance	4	3	7	10
Orthoptics	7 (58%)	3 (16%)	4 (10%)	1 (7%)
Total No. patients	12	19	39	14

^{*}Arithmetic mean.

caused by decreased accommodative effort as a result of correction of additional hypermetropia.

In all patients in Group 4, function deteriorated because of an increase of the near deviation, which resulted in eventual loss of fusion control despite maximal bifocal correction. Atropine refraction was performed at semiannual intervals as in the other groups, and optical undercorrection of an increasing hypermetropia was not a contributing factor. Four patients in Group 4, in whom deterioration of binocular functions at near fixation was diagnosed, were treated with miotics as well as bifocals; however, this therapy was ineffective inffurther reducing the near deviation by a significant amount.

DISCUSSION

Thirty-one of 84 patients were either cured or improved in a manner suggesting eventual cure with further therapy and an additional 39 patients maintained fusion with bifocals for several years. Thus, bifocals emerge as a powerful and effective tool in the management of esotropia with a high AC/A ratio.

In patients in whom fusional control deteriorated under therapy (Group 4), AC/A ratios were generally lower at the beginning of therapy than in those who did well. This observation suggests there are two types of esotropia which share the common characteristic of having a deviation that is significantly greater at near than at distance fixation. The first type is more frequently encountered in patients whose deviation decreases with bifocal therapy; the AC/A ratio is high and the near deviation can be effectively reduced with additional plus lenses. The second type of esotropia is most commonly seen in patients in Group 4; the AC/A ratio is normal or lower than normal, and bifocals are less effective in reducing the near deviation. Our study has shown that in

[†]Includes esophorias and esotropias.

patients with the first type of esotropia, for which we suggest the term "accommodative convergence excess," the AC/A ratio and, thus, the near deviation tend to decrease with time so that the bifocals can be gradually reduced and eventually discontinued. Orthoptic therapy in such patients has contributed to good functional results. With the second type, excessive convergence must occur on a basis other than accommodation, perhaps from increased tonic convergence, for relaxation of accommodation by additional plus lenses had little effect on the deviation. We suggest the term "nonaccommodative convergence excess" for this type. Our study has shown that patients with this anomaly, while initially responding well to bifocals as long as the near deviation is small, will eventually lose fusional control as the near deviation increases beyond a point where additional plus lenses are effective. Thus, a low AC/A ratio at the beginning of biocal therapy emerges as an unfavorable prognostic sign, and the physician must guard against deterioration of binocular function when such patients are treated with bifocals.

The question arises as to how to treat patients of the Groups 3 and 4 type. Those who depend on their bifocals to maintain fusion can be observed for many vears in the hope that fusional control will eventually improve and that bifocal power can be reduced. However, treatment in many cases is merely symptomatic and once a child reaches his teens bifocals may become a cosmetic problem and may interfere with athletic activities. Rather than keeping such children in bifocals, we have recently begun to recess both medial recti when such patients reach their early teens. The initial results have been encouraging, but follow up over a longer period of time is necessary to assess the ultimate value of this therapy. On the other hand, those patients who lose their ability to fuse at near while under maximal bifocal therapy (Group 4)

deserve surgical correction without delay to preserve binocular functions.

SUMMARY

We treated 84 patients with a partially refractive accommodative esotropia with bifocals. Twelve patients were able to fuse without bifocals at the end of therapy; in 19, the bifocal power could be reduced and further improvement can be expected in the future. Thirty-nine remained dependent on bifocals; and in 14, fusion had deteriorated in spite of therapy. Patients with a high AC/A ratio and those receiving supportive orthoptic treatment seemed to fare best with bifocals. In those with a low AC/A ratio, fusion tended to deteriorate because of a slowly increasing esodeviation at near fixation.

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SECONDARY DIABETIC RETINOPATHY IN CHRONIC PANCREATITIS

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Although many reports have been done on diabetic retinopathy, only a few studies have been done on secondary diabetic retinopathy because of its low incidence. In cases of chronic pancreatitis, diabetes is caused by the disturbance of pancreatic endocrine functions. Diabetic retinopathy has been widely investigated, since the analysis of a disease such as pancreatic diabetes is useful in elucidating the mechanism of diabetes mellitus. Additionally, retinopathy caused by pancreatic diabetes may also provide information useful in explaining the mechanism of retinopathy in primary diabetes.

We investigated retinopathy in pancreatic diabetes to compare primary and secondary diabetic retinopathy.

MATERIAL AND METHODS

The 47 cases we studied were diagnosed as chronic pancreatitis by the pancreatic function test or by a survey of surgery over a ten-year period (1966 to 1975) at the Diabetes Mellitus Clinic of Tohoku University. There was no family history of diabetes. The eyes of the patients were examined in detail by direct and indirect ophthalmoscopy. Fluorescein angiography was performed in 11 cases.

Before surgery, we performed the secretin-pancreozymin test in all patients. Lipid content in the stool was measured in patients postoperatively. The patients were divided into four groups according to the grade of decrease in pancreatic exocrine function: normal, slight, moderate, and severe. Furthermore, patients underwent standard oral glucose tolerance test with 50 g of glucose. The patients were then divided into three groups: normal, borderline, and diabetic. Glucose tolerance was classified according to the recommendation of the Committee of the Iapan Diabetic Society. Diabetic glucose tolerance was defined as the peak level of blood glucose of more than 180 mg/100 ml, and as the two-hour level of more than 140 mg/100 ml. To investigate the function of the islets of the pancreas, we studied plasma response of insulin and glucagon to arginine.¹

In all cases diagnosed as pancreatitis, patients fulfilled the following criteria: (1) typical episodes of acute pancreatitis, (2) calcified pancreas from X-ray film findings, and (3) abnormalities of the pancreozymin-secretin test (normal values: amylase value, 1.6 ml/kg of body weight; bicarbonate value, 70 mEq/liter; and serum amylase level, 800 units/kg of body weight).

RESULTS

According to the results of the glucose tolerance tests, the patients were divided into three groups: two were normal, ten borderline, and 35 diabetic (Table 1). The age distribution of retinopathy appears in Table 2. The peak ages of occurrence of retinopathy were 30 to 40 years. In seven of 28 cases of pancreatic diabetes, diabetic retinopathy developed within ten years of the onset of diabetes

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TABLE 1
RESULTS OF GLUCOSE TOLERANCE TEST
AND OCCURRENCE OF RETINOPATHY
IN CHRONIC PANCREATITIS

			Glucose	
			Folerance	
			Test	
	No. of	Diabetic*	Border-	Normal
	Cases		line	
Men	38	30 (7)	7	1
Women	9	5(1)	3	1
Total	47	35 (8)	10	2

^{*}Numbers in parentheses indicate occurrence of retinopathy.

mellitus (Table 3). Calcified pancreatitis occurred in seven of eight cases with retinopathy, and chronic noncalcified pancreatitis occurred in one of eight cases after several attacks of pancreatitis (Table 4).

Retinopathy with several microaneurysms was found in seven cases by direct ophthalmoscopy, but only one case confirmed by fluorescein angiography revealed a few microaneurysms, dilatation of precapillary venules, and leakage of the dye from the capillary bed. In three cases, dye leakage was almost always observed at the same area of microaneurysm (Figure). These hyperfluorescent dots were aneurysms and not drusen. The grade of retinopathic changes in all cases was limited within grade Ia to IIa (that is, microaneurysm to dot hemorrhage), ac-

TABLE 2
SEX, AGE, AND INCIDENCE OF PATIENTS WITH RETINOPATHY OF PANCREATIC DIABETES

Age (yrs)	Men	Women	Incidence of Retinopathy
20-29	1	2	1
30-39	12	2	3
40-49	15	1	2
50-59	7	0	2
60-69	3	2	0
70-	1	1	0
Total	39	8	8

cording to Scott's classification² in primary diabetic retinopathy. Thus, the slight diabetic retinopathy observed in this study is of particular interest.

Of nine cases showing normal lipid

TABLE 3
RETINOPATHY IN RELATION TO DURATION OF DIABETES

Duration of Diabetes (yrs)	No. of Patients	No. of Cases of Retinopathy
0-5	15	5
5-10	10	2
10-15	2	1
Over 15	1	0
Unknown	7	0
Total	35	8

contents in the stool postoperatively, retinopathy was found in two cases. Retinopathy was also observed in four of nine cases with mild disturbance of pancreatic

 ${\bf TABLE~4}$ Clinical data of patients with diabetic retinopathy in chronic pancreatitis*

								Pancreatic Endocrine Function		
Case No.	Sex	Age (yrs)	Family History of Diabetes	Duration of Pancreatic Diabetes	Grade of Diabetic Retinopathy	Pancreato- lithiasis	Pancreatic Exocrine Disturbance	Diabetic Glucose Tolerance	Insulin Response	Glucagon Response
1	F	26	400	7 vrs	Scott Ia	Present	Moderate	Slight	N.E.	N.E.
2	M	36	****	7 vrs	Scott IIa	Present	Moderate	Moderate	N.E.	N.E.
3	M	56		9 vrs	Scott Ia	Absent	Severe	Severe	N.E.	N.E.
4	M	36	-	4 yrs	Scott Ia	Present	Moderate	Moderate	Decreased	Decreased
5	M	37		4 vrs 5 mo	Scott Ia	Present	Normal	Slight	Decreased	Normal
6	M	45		12 yrs 1 mo	Scott Ia	Present	Severe	Slight	N.E.	N.E.
7	M	55		5 yrs	Scott Ia	Present	Normal	Slight	Decreased	Decreased
8	M	44	****	9 mo	Scott Ia	Present	Moderate	Severe	N.E.	N.E.

^{*}N.E. indicates no examination.

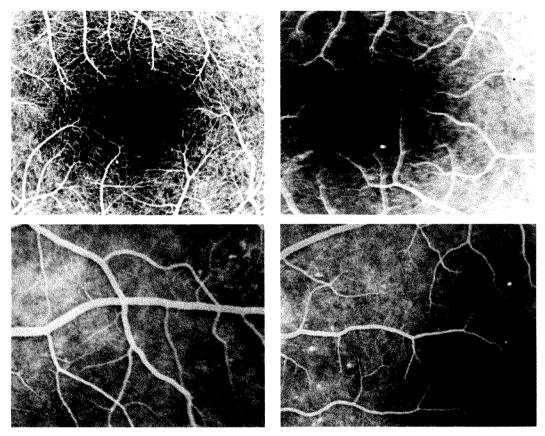


Figure (Maekawa and associates). Fluorescein fundus angiograms of diabetic rectinopathy secondary to chronic pancreatitis (top left, Case 5; top right, Case 6; bottom left, Case 7; bottom right, Case 8 of Table 4).

exocrine function, and in two of 18 cases with severe exocrine disturbance. However, we observed no relationship between retinopathy and disturbance of pancreatic exocrine function (Table 5).

All cases of diabetic retinopathy showed an abnormal glucose tolerance curve. The arginine tolerance response test was performed in 13 patients. Plasma responses of both insulin and glucagon to arginine were decreased in ten of 13 cases. Two of 13 cases showed decreased insulin and increased glucagon response. The arginine tolerance response test was performed in three of eight cases with diabetic retinopathy and showed a decrease in insulin and glucagon response,

and normal glucagon response in one case (Table 4).

DISCUSSION

In 1963 Marks and Bank³ observed abnormal glucose tolerance in 71% of chronic pancreatitis cases, whereas Matsuda⁴ observed the same in 89% of cases including borderline and diabetic types. We observed abnormal glucose tolerance curves in 74% of the diabetic cases and in 94% of the borderline and diabetic cases.

Sprangue⁵ first reported diabetic retinopathy in two cases with secondary diabetes caused by pancreatitis. The retinopathy appeared in the fundus ten years

TABLE 5
RETINOPATHY IN RELATION TO DISTURBANCE
OF PANCREATIC EXOCRINE FUNCTION

Pancreatic Exocrin	ne	No. of Cases of
• Function	No. of Patients	Retinopathy
Normal*	9	2
Disturbance		
Slight	5	0
Moderate	9	4
Severe	10	2
No performance	6	O

^{*}Postoperative case.

after the onset of pancreatic diabetes. Deckert⁶ reported a case of retinopathy that was detected 12 years after the onset. Duncan, Macfarlane, and Robson⁷ reported a case of retinopathy that appeared 24 years later, and Tutin and associates,8 three cases after nine, 13, and 18 years, respectively. However, these reports lack a detailed description of the retinopathy in secondary diabetes. Fukuda and Yamazaki9 reported a case of progressive diabetic retinopathy that manifested itself ten years after the onset of diabetes. Their patient died of severe nephropathy. Koh¹⁰ reported a case that appeared seven years later. Recently, Sevel, Bank, and Jackson¹¹ reported two cases of diabetic retinopathy in 27 cases of chronic pancreatitis. A few microaneurysms were recognized by fluorescein angiography in one case. The incidence of diabetic retinopathy in the control group of patients with primary diabetes mellitus was 30%, whereas the incidence in the pancreatic diabetes group was 7.4%.

In our study, 35 of 47 cases with chronic pancreatitis showed diabetic characteristics with respect to the glucose tolerance curve, and moreover, diabetic retinopathy was found in eight cases (23%) of pancreatitis with diabetic glucose tolerance. This percentage was higher than in the

previous reports. The reason for such a high incidence of diabetic retinopathy may be the use of Luorescein angiography; the possibility of pancreatic diabetes occurring in primary diabetes must also be considered. Moreover, a comparison between pancreatic diabetes and primary diabetes is difficult because of the few published reports on pancreatic diabetes. As reported by Sevel, Bank, and Jackson,11 the incidence of retinopathy in secondary diabetes was still lower than that of retinopathy in primary diabetes.¹² They speculated that the low incidence results because the majority of patients with pancreatic diabetes die at a fairly early age and do not allow enough time for observation. Generally, retinopathy in secondary diabetes occurs after a long duration of diabetes. However, retinopathy was found within five years of the onset of secondary diabetes in five of our cases. The chronic pancreatitis was probably in an advanced stage, which suggests a long duration of diabetes. With the exception of Fukuda and Yamazaki's case,9 which might be a mixed type having a genetic factor of primary diabetes, fundus changes in almost all reported cases and in our cases of pancreatic diabetes showed slight retinopathy.

Recently, it has been suggested that pancreatic diabetes is protected against retinopathy either because of a relatively deficient growth hormone level¹³ or a relative hypolipidemia.¹⁴

The mechanism of diabetic retinopathy has been discussed mainly on the basis of pancreatic endocrine function, especially the insulin response. But Unger and Orci¹⁵ reported that diabetes mellitus may not be the simple consequence of relative or absolute deficiency of insulin alone, but may be related to the presence of glucagon. They hypothesized that diabetes mellitus is the consequence of a bihormonal disorder in which a relative

or absolute deficiency of insulin and a relative or absolute excess of glucagon are both etiological factors, and that the presence of glucagon is essential to diabetes mellitus. From the results obtained in this study, we think that the dysfunction of metabolism in pancreatic diabetes is somewhat different from that in primary diabetes. Previously, we reported that insulin and glucagon responses were reduced in chronic pancreatitis as shown in the arginine test, whereas primary diabetes showed decreased insulin response and increased glucagon responses. Therefore, the metabolic conditions of primary and pancreatic diabetes seem to be different. The arginine test was performed in 13 cases with chronic pancreatitis, since diabetic retinopathy might be caused by the endocrine dysfunction of the pancreas. Three cases with retinopathy among 13 cases of chronic pancreatitis did not show the same pattern of pancreatic endocrine dysfunction characteristic of primary diabetes cases. Two of the three cases with retinopathy showed reduced insulin and glucagon responses, and one of the three cases showed decreased insulin and normal glucagon responses. The insulin: glucagon ratio was markedly decreased in primary diabetes and was almost normal in pancreatic diabetes with chronic pancreatitis. But it is not certain whether our cases showed the same pattern of endocrine function of the pancreas as in primary diabetes because we could not observe the endocrine function of the pancreas for a long enough time in such patients.

Although pancreatic endocrine functions in Cushing's syndrome have the same pattern as in primary diabetes, ¹⁶ which showed decreased insulin response and increased glucagon response, retinopathy was rarely observed in Cushing's syndrome. ¹⁷ Thus the reason for the low incidence of retinopathy in secon-

dary diabetes cannot be explained only by the abnormalities in metabolism and by deviation of insulin:glucagon ratio.

Matsuda⁴ reported that chronic pancreatitis frequently causes abnormal glucose tolerance. However, the results obtained in this experiment indicate there is no relation between the frequency of diabetic retinopathy and the exocrine dysfunction of the pancreas.

The disturbance of glucose tolerance occurs frequently in patients with chronic pancreatitis, but its degree is not so remarkable as in primary diabetes. Moreover, it is apparent that various factors, such as genetic factor, age, diabetic duration, and the control of blood glucose may be responsible for diabetic retinopathy. Since all the cases in our study have no familial history of diabetes mellitus, we suggest that they have no genetic factor for primary diabetes in these cases. According to Siperstein, 18 the capillary basement membrane of the muscle showed distinct morphological changes in primary diabetic patients compared with those in secondary diabetic ones. In primary diabetes, the width of the capillary basement membrane was measured to be about 240 nm, whereas in secondary diabetes of chronic pancreatitis, it was a little narrower than 160 nm, similar to the width, 110 nm, of normal controls. The results suggest that hyperglycemia due to conditions other than genetic diabetes is not likely to produce typical retinopathy. Therefore, the low frequency of diabetic retinopathy in secondary diabetes may be related to the lack of a diabetic genetic factor. On the other hand, diabetic retinopathy might occur in cases with a long duration of glucose intolerance.

SUMMARY

We found abnormal glucose tolerance curves in 45 of 47 patients with chronic pancreatitis and observed secondary diabetic retinopathy in eight of 45 cases showing slight changes in the fundus. Abnormal glucose tolerance curves were somewhat related to the exocrine dysfunction of the pancreas. Slightly abnormal glucose tolerance curves were observed frequently in patients with chronic pancreatitis, and both insulin and glucagon responses were decreased. We could not explain the cause of the low frequency of secondary diabetic retinopathy in pancreatic diabetes from the results of insulin and glucagon response tests.

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THE ULTRASTRUCTURE OF THE RETINA IN ADULT METACHROMATIC LEUKODYSTROPHY

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Metachromatic leukodystrophy of the late infantile and adult types chiefly affects the myelin sheaths of the central and peripheral nervous system. Accumulation of residual bodies has also been demonstrated in neurons by light microscopy² and by electron microscopy.3 In the late infantile form of metachromatic leukodystrophy,4 metachromatic granules have been documented in the retina, both by light microscopy^{5,6} and electron microscopy.7 Since lysosomal disorders of late clinical onset tend to reveal a less widespread morphological involvement of cells and organs lysosomal pathology in the retina of adult metachromatic leukodystrophy may differ from that of the infantile type. The following studies, to our knowledge, represent the first account on the ultrastructure of the retina in adult metachromatic leukodystrophy.

CASE REPORT

This 46-year-old woman first complained of backache at the age of 40 years. Only a mild facial hypokinesia was noted. Motor nerve conduction velocities of ulnar and peroneal nerves were reduced to 36 m per second and 30.9 m per second, respectively. Two years later she developed depression. She walked with small steps and flexed knees; her gait and posture were unsteady. At 44 years of age, she had dementia, dysarthria, a mild central facial palsy, and a Babinski sign on the left side. Both pupils reacted equally and sluggishly to light. A slight nystagmus to the left was noted. Retinal and papillary changes were absent. Her sural nerve conduction velocity was 23.1 m per second. Her

physical and mental state continued to deteriorate. Shortly before her death, ophthalmoscopy only revealed slightly pale optic disks. No further ophthalmopathological findings were recorded. Biochemically, her leucocytic arylsulfatase A activity had shown values of 2.8 and 5.7 nanomoles of nitrocatechol per hour per milligram of protein (normal, 50 to 125). She had excreted 0.2 to 0.4 and 0.4 mg/24 hours of sulfatides on two occasions (normal, less than 0.01). Her brother and her son were homozygously afflicted with adult metachromatic leukodystrophy. Detailed reports on her clinical and laboratory findings have previously been published.^{8,9}

MATERIAL AND METHODS

Posterior halves of both eyes were removed within three hours after death. Parts of both retinas were frozen in isopentane in liquid nitrogen, cryosectioned and stained with hematoxylin-eosin, acid cresyl violet, toluidine blue, PAS, and for acid phosphatase. Formalin-fixed, paraffin-embedded sections of both optic nerves were stained with hematoxylineosin, luxol fast blue-PAS, the Klüver-Barrera technique, and the Bodian method. Additional small blocs of tissue were fixed in cacody-late-buffered 3 % glutaraldehyde and further processed for electron microscopy by using previously outlined techniques.10

RESULTS

Light microscopy—Cryostat sections of the retina did not reveal metachromatic material in any retinal layer or the underlying choroid. Acid phosphatase activity was prominent in ganglionic cells and pigment epithelial cells (Fig. 1, top left). One-micron thick, Araldite-embedded, toluidine blue-stained sections showed a regular layering of the retina with wellpreserved photoreceptors. The pigment epithelial layer was studded with brown and black pigment granules. Several gan-

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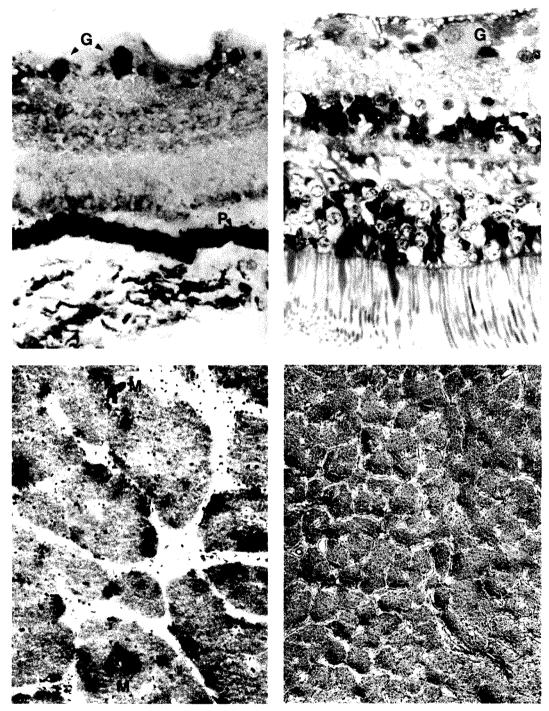


Fig. 1 (Goebel and associates). Top left, Acid phosphatase activity in pigment (P) epithelial layer and in ganglionic (G) cells. Top right, Well-preserved layers of the retina with a large ganglionic (G) cell. Bottom left, Metachromatic (M) granules in optic nerve. Bottom right, Regular myelination and glial cells in optic nerve (top left, $\times 300$; top right, araldite, toluidine blue, $\times 450$; bottom left, acid cresyl violet, $\times 120$; bottom right, LFB-PAS, $\times 50$).

glionic cells revealed numerous, cytoplasmic inclusion bodies (Fig. 1, top right). Metachromatic material (Fig. 1, bottom left) and acid phosphatase activity were encountered in optic nerve cryostat sections. Paraffin-embedded optic nerves did not disclose any clear-cut demyelination (Fig. 1, bottom right) or loss of axons.

Electron microscopy—Abnormal lysosomes were found in only a few ganglionic cells (Fig. 2). These residual bodies were bound by a trilaminar membrane. They varied considerably in size and fine structural detail. Intralysosomal membranes were occasionally arranged in small stacks (Fig. 3, top). Other residual bodies contained electronlucent areas, faint interspersed membranous profiles, and single or multiple round opaque droplets, probably representing neutral lipids (Fig. 3, middle left). In other residual bodies, faintly visible membranes alternated with electronlucent, slit-like spaces and a finely granular dark matrix (Fig. 3, middle right). A few large inclusions showed a conglomerate of membranes, granular matrix, lipid droplets and stacks of lamellae loosely arranged in a circular fashion (Fig. 3, bottom). There were many transitions between membranous residual bodies and regular lipofuscin. Only ganglionic cells harbored such membranous residual bodies. Large lipofuscin granules composed of a granular matrix and various lipid droplets were occasionally found in other nerve cells of the retina, such as the bipolar layer, processes of the inner plexiform layer, glial cells, and various mural cells of the vasculature. The photoreceptors were well arranged. In several photoreceptor inner segments, usually closely related to the external limiting membrane, we found single or multiple small membranebound inclusions (Fig. 4); higher magnification revealed that these consisted of randomly arranged, ill-defined membranous fragments (Fig. 4, inset). These bodies differed from regular mitochondria of photoreceptor inner segments. A matrix and mitochondrial cristae could not be identified in these bodies. The pigment epithelial cells were well endowed with melanin-bearing inclusions and other granular residual bodies, apparently representing lipopigments. In some such lipopigment bodies, wedge-shaped melanin granules still exhibited a faint striatal skeleton of melanosomal origin. Bruch's membrane was occasionally broadened by extracellular amorphous and vesicular material located between the basal lamina of the pigment epithelial cells and the basement membrane of the choriocapillaris. The choroid revealed many melanocytes that harbored only melanin inclusions and no lipofuscin or other abnormal residual bodies. Macrophages of the choroid contained lipopigments and a few huge, electronlucent vacuoles with only marginal, finely granular, electron-dense material (Fig. 5, left). Other macrophages showed large, membrane-bound residual bodies whose content consisted of flecks of granular matrix, multiple lipid droplets, and numerous flame-shaped melanin bodies (Fig. 5, right). Lipofuscin was encountered in Schwann cells of unmvelinated axons of the choroid. Membranous residual bodies (Fig. 6) were present in Schwann cells of choroidal myelinated axons that resembled inclusions in sural nerve Schwann cells of this patient.11

Discussion

Clinically and ophthalmologically, involvement of the retina by the sulfatide storage process did not become apparent. Slight pallor of both optic disks was only seen shortly before death. In late infantile metachromatic leukodystrophy, a cherry red spot may occasionally appear. ¹² In this respect, ophthalmoscopic



Fig. 2 (Goebel and associates). A ganglionic cell is filled with numerous residual (R) bodies, some of which show a globular (G) substructure $(\times 14{,}700).$

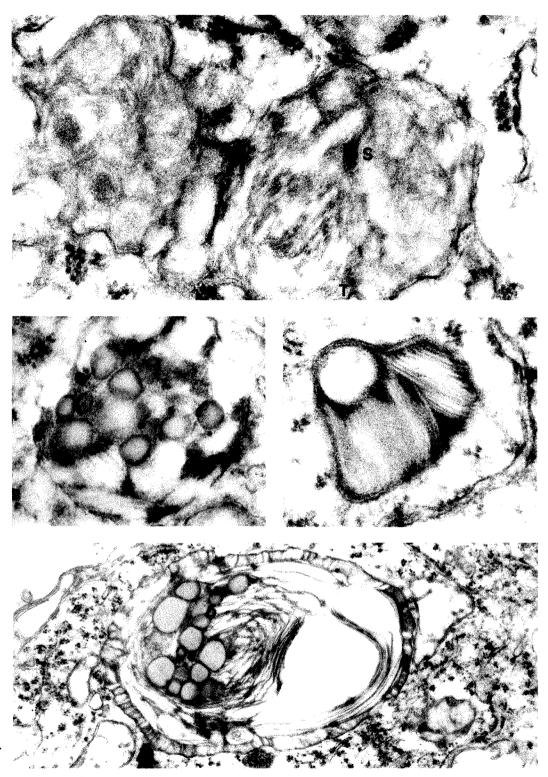


Fig. 3 (Goebel and associates). Several residual bodies of ganglionic cells at higher magnification. Top, Only a few membranes, forming small stacks (S), are encountered. T, trilaminar limiting membrane. Middle left, In addition to membranes and granular matrix, several round opaque lipid globules are present in this lysosome, Middle right, A granular matrix alternates with stacks of faintly visible membranes. Bottom, A conglomerate body contains numerous membranes, lipid droplets, and a peculiar peripheral arrangement of membranes (top $\times 66,700$; middle left, $\times 72,500$; middle right, $\times 72,500$; bottom $\times 24,000$).

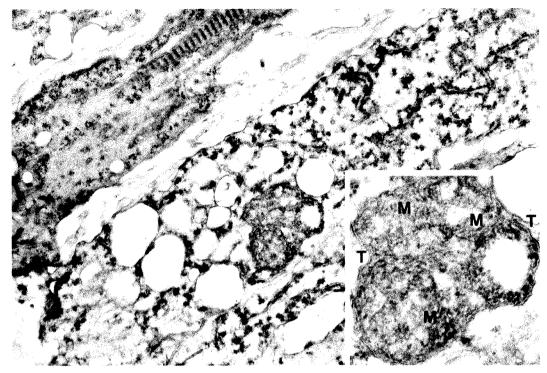


Fig. 4 (Goebel and associates). A photoreceptor inner segment contains a body, which at higher magnification (inset) consists of numerous short membranes (M), surrounded by a trilaminar (T) membrane $(\times 18,400;$ inset, $\times 43,500)$.

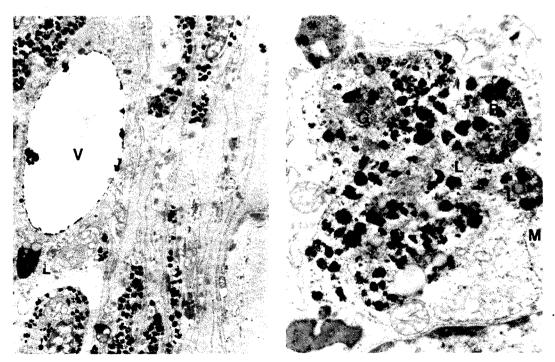


Fig. 5 (Goebel and associates). Left, A choroidal macrophage contains a huge membrane-bound vacuole (V) with marginal electron-dense material and a lipopigment granule (L). Right, A large membrane-bound (M) residual body of a choroidal macrophage contains granular material (G), lipid droplets (L) and numerous minute melanin bodies (B) (left, $\times 3,800$; right, $\times 12,800$).

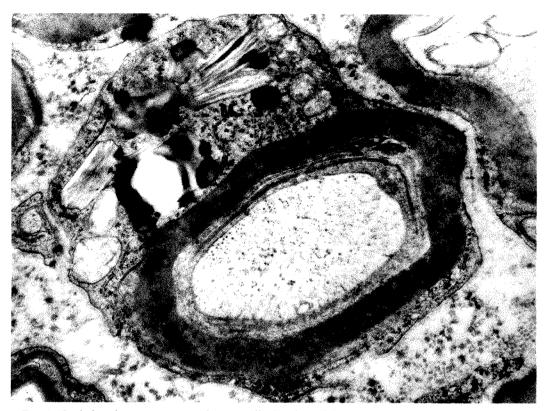


Fig. 6 (Goebel and associates). A Schwann cell of a choroidal myelinated axon contains membranous residual bodies (M) and lipid droplets (L) $(\times 18,400)$.

findings of the retina in metachromatic leukodystrophy resemble those seen in the GM_1 gangliosidoses, which reveal a cherry-red spot in about 50% of the patients afflicted with Type I, but not in the Type II patients, ¹³ although membranous residual bodies do accumulate in the retina in GM_1 gangliosidosis, Type II. ¹⁴

In late infantile metachromatic leukodystrophy, metachromatic material accumulates in several layers of the retina such as ganglionic and pericapillary cells,⁷ the inner plexiform layer,⁵ the internal granular and neuroepithelial layers⁶; but in adult metachromatic leukodystrophy, accretion of sphingolipids appears to be confined to the ganglionic cells. The reason for the sole involvement of ganglionic cells in adult metachromatic dystrophy is not clear.

There are similar lysosomal entities of

late onset with less accretion of residual bodies than the respective forms of early onset.15 In Gaucher's disease, only the infantile type reveals neuronal involvement. 16 Other such examples are Type II glycogenosis¹⁷ and GM₁ gangliosidoses.¹³ Seemingly, the degree of reduced arylsulfatase A activity in metachromatic leukodystrophy does not correlate with the amount of stored sulfatides or with the clinical severity of the disease.18 The accretion of membranous residual bodies in ganglionic cells of the retina in adult metachromatic leukodystrophy may also reflect subtle damage to distal parts of their axons because of the myelin pathology. Or it may indicate a sulfatide metabolism different from that of other neuronal cells in the retina. There may then be classes of retinal and central nervous system neurons that show a sulfatide metabolism resulting in sulfatide accumulation, such as anterior horn cells³ or ganglionic retinal cells, and classes of other neurons that do not, such as cerebral and cerebellar³ or other retinal nerve cells. This presents a challenge to the biochemist to study the sulfatide metabolism in various classes of neurons in metachromatic leukodystrophy.

Residual bodies considered typical of metachromatic leukodystrophy, such as prismatic¹⁹ or tufaceous inclusions,¹ were not found in the retina of this patient, although they were present in her sural nerve.¹¹ Apparently, in metachromatic leukodystrophy, the ultrastructural morphology of intraneuronal residual bodies differs from that of glial and mesenchymal cells. The bodies have been described as laminated bodies in spinal anterior horn neurons,³ as laminated inclusion bodies in retinal neurons, and even as similar to residual bodies of Tay-Sachs disease.⁴

Appearance of lipofuscin increases with age.20 This is probably the reason for the many lipopigment granules observed in retinal cells of our patient. The acid phosphatase activity noted by light microscopy apparently is caused by these lipopigments in pigment epithelial cells, as well as to the membranous residual bodies of ganglionic cells. The process of lipofuscin generation, localized in the lysosomal compartment, may be enhanced by a superimposed lysosomal disease, such as metachromatic leukodystrophy. In metachromatic leukodystrophy, this may result in the formation of residual bodies that contain both lipofuscin and leukodystrophy-specific metachromatic material. A similar combined occurrence of lipofuscin and lysosomal glycogen has been observed in infantile Type IIglycogenosis.21

Although metachromatic material was evident in both optic nerves, no overt

demyelination and axonal loss were encountered. This is reflected by the mild pallor of both optic disks, noted shortly before the patient's death. In another case of adult metachromatic leukodystrophyr,²² the optic nerves also had been exempted from the otherwise advanced demyelinating process.

SUMMARY

A 46-year-old woman afflicted with biochemically proven metachromatic leukodystrophy had only mild optic atrophy shortly before her death. Repeated earlier ophthalmoscopic examinations had not revealed any retinal abnormalities. Light microscopy of the retina showed strong acid phosphatase activity in both enlarged ganglionic cells and pigment epithelial cells. Demyelination of both optic nerves was not noted. Ultrastructurally, membranous lysosomal residual bodies were confined to ganglionic cells. We found lipofuscin material in pigment epithelial cells, but also within metachromatic leukodystrophy-specific residual bodies of ganglionic cells. The presence of lipofuscin represents the "wear-andtear" phenomenon, possibly enhanced by the metachromatic leukodystrophy.

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TERATOID INTRAOCULAR MEDULLOEPITHELIOMA

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Tumors arising in the neuroectodermal epithelium of the ciliary body are rare.1 Zimmerman² has studied these epithelial tumors extensively and classified³ them into two main groups: (1) the medulloepitheliomas occurring in children, which are often termed diktyomas⁴⁻⁶; and (2) the acquired adenomas and adenocarcinomas. He believes that the intraocular medulloepitheliomas are congenital neoplasms; or that at least the anlage from which they arise is present at birth, even though the clinical manifestations that lead to their first recognition may not develop until long after birth. In these uncommon congenital tumors, heteroplastic elements, not usually observed in the eve, such as brain tissue, cartilage, as well as striated muscle fibers, usually resembling moderately to well-differentiated rhabdomyoblasts, have been described.3,7

The purpose of this report is to describe and illustrate our light microscopy observations in a similar tumor, a teratoid malignant medulloepithelioma at an advanced stage of its growth.

MATERIAL AND METHODS

The eye fixed in formalin was sent to us from Liberia for consultation. It had been enucleated from a 1-year-old black girl suffering from severe pains; examination revealed a blind right eye, tactilely hard, slightly proptosed, and containing an intraocular tumor. The preoperative diagnosis was retinoblastoma. The fellow eye was normal.

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The bulb measured 20 mm in sagittal diameter and 18 mm in transverse and vertical diameters. The optic nerve was cut immediately at its exit.

The eye, when divided sagittally into two halves through the optic disk, revealed a huge neoplastic mass that almost filled the vitreous cavity (Fig. 1); the mass had destroyed and replaced the normal architecture of the retina, ciliary body, and iris. Paraffin-embedded and frozen sections were prepared from the right and left half, respectively.

Sections prepared from the paraffin blocks were stained with hematoxylin and eosin, Verhoeff-van Gieson's stain, Gomori's trichrome, phosphotungstic acid-hematoxylin and with Alcian blue (with and without prior treatment with hyaluronidase). Frozen sections were

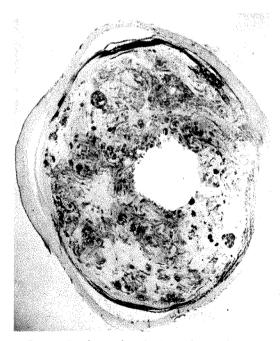


Fig. 1 (Yanko and Behar). Midsagittal section through the globe showing extension of tumor (hematoxylin and eosin, \times 5).

stained with the PAS technique, with and without prior treatment with malt diastase, and with the panoptic technique of Del Rio Hortega.

RESULTS

The neoplasm varied markedly in its microscopic appearance. It consisted of aggregations, sheets and cords, or moderately large, rounded, or elongated but otherwise undifferentiated cells. Many of these cells appeared anaplastic and had mitotic figures. Adjacent to the areas of undifferentiated cells were other structures that differed greatly in their histologic appearances: tubular elements lined by multilayered and poorly differentiated neuroepithelial cells that closely resembled the medullary epithelium of the embyonic retina (Fig. 2); rosette-like configurations analogous to those of retinoblastoma; and bundles of spindle-shaped and strap-like cells abundant with eosinophilic cytoplasm rich in glycogen. The Hortega technique demonstrated beautifully the typical cross striation of rhabdomyocytes in many of these (Fig. 3). Various neoplastic cells were also observed as a diffuse and massive infiltration throughout the optic nerve. Islands of hyaline

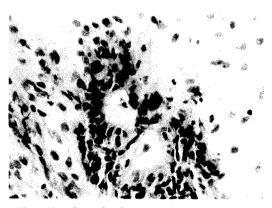


Fig. 2 (Yanko and Behar). Tabular structures lined by poorly differentiated cells, imitating neural tube (hematoxylin and eosin, \times 320).

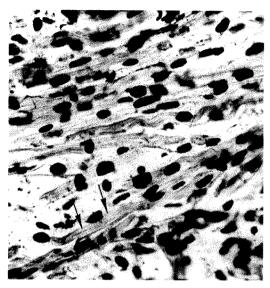


Fig. 3 (Yanko and Behar). Rhabdomyoblasts with a large hyperchromatic and centrally located nucleus and cytoplasm with both longitudinal and cross striations (Del Rio Hortega panoptic silver impregnation technique, × 420).

cartilage surrounded by bundles of the strap-like cells were also present. This cartilaginous tissue contained a matrix that stained positive with Alcian blue and resisted digestion with hyaluronidase. Most of the tumoral elements were embedded in a mucoid matrix which stained positive with Alcian blue, but was sensitive to hyaluronidase. Noticeable throughout the periphery of the tumoral mass were numerous areas of calcification, apparently involving necrotic tumor tissue (Fig. 4).

DISCUSSION

Teratomas, etymologically "monstrous tumors," are lesions which contain an assemblage of organoid tissues mostly foreign to the site in which they arise. Verhoeff⁸ in 1904 described in minute detail a rare intraocular tumor that arose from the nonpigmented ciliary epithelium. He called it teratoneuroma. He pointed out that the vitreous humor observed within the lesion was newly formed and

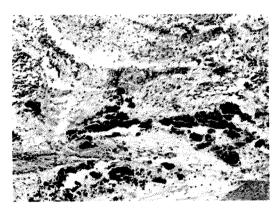


Fig. 4 (Yanko and Behar). Foci of calcification in necrotic areas of tumor. (hematoxylin and eosin, \times 105)

represented an integral part of the tumor. He also believed the formation of vitreous indicated the presence of mesoblastic tissue in this neuroectodermal tumor and, accordingly, related the case to the teratoma class of neoplasms. In reviewing this case, Zimmerman³ found a total resemblance of Verhoeff's tumor to a "pure" medulloepithelioma. Obviously Verhoeff had inappropriately used the term "terato-neuroma" for the tumor he described.

According to Zimmerman,3 the pure medulloepitheliomas comprise elements that closely resemble the medullary epithelium and occasionally are made up of additional structures resembling those derived from the secondary optic vesicle. such as retinal pigment epithelium, ciliary epithelium, vitreous, and neuroglia. The malignant type of this tumor also contains masses of neuroblastic cells with numerous mitotic figures resembling somewhat undifferentiated retinoblassome Additionally, Flexnertoma. Wintersteiner rosettes may also be present.

The teratoid type of medulloepithelioma contains heteroplastic tissues or tissue elements, as well as the medulloepitheliomatous components. Of these by far the most frequently observed element is hyaline cartilage.⁵ Zimmerman has described skeletal muscle in the tumor that usually resembles moderately to well-differentiated rhabdomyosarcomas.^{3,7} The presence of striated muscle elements in cerebelar medulloblastomas has long been recognized,^{9,10} and recently Woodruff and associates¹¹ added three new cases to the seven previously reported cases of peripheral nerve tumors containing rhabdomyosarcoma. These are referred to by the same authors as malignant triton tumors.

The histological observations made in our case are similar to those reported in ocular teratoid medulloepithelioma. Although it was recognized, clinically, when the patient was 1 year of age, it had extended and reached an advanced stage and spread throughout the optic nerve. Even though the occurrence of an intraocular tumor of this kind is rare, one should keep it in mind when proptosis and blindness are associated with a painful eve in childhood. The calcification observed in our case indicated that this clinical finding may not always be pathognomonic of retinoblastoma, 12 as is generally believed.

SUMMARY

The right eye of a 1-year-old black infant with a painful blind eye was studied histologically. It contained a tumor at an advanced stage of growth. The tumor consisted of moderately large, round, and elongated undifferentiated cells and anaplastic cells with mitotic figures combined with multilayered, poorly differentiated epithelial cells that resembled the medullary epithelium of embryonic retina. Strap cells with cross striation typical of striated muscle were also present, together with islands of hyaline cartilage and areas of calcification.

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OPHTHALMIC MINIATURE

The glance she gave him was almost hostile, and curiously hard; for her face could look hard sometimes, in spite of its youth and its flower-like skin. Those modish tortoise-shell spectacles gave her a very self-possessed look. Spectacles are queerly expressive things—almost more expressive, indeed, than eyes.

George Orwell, *Burmese Days*New York, Signet, 1963

ASSOCIATION OF PRESUMED OCULAR HISTOPLASMOSIS WITH HLA-B7

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The best examples of the associations of certain HLA antigens with several specific diseases involving the eye are the high correlations between HLA-B27 and ankylosing spondylitis1 and Reiter's syndrome.2 Both of these diseases have a high incidence of acute recurrent iridocyclitis. HLA-B8 has been linked with the keratitis sicca in Sjögren's syndrome^{3,4} and with myasthenia gravis.⁵ Sjögren's syndrome has been linked with HLA-DW3.6 Vogt-Koyanagi-Harada's disease has been associated with HLA-BW221.7 Additional studies have recently reported an increased frequency of HLA-B12 and B7 with primary open-angle glaucoma8 and HLA B13 and BW17 in the pigment dispersion syndrome.9 We report herein a correlation between the HLA type B7 and the clinical syndrome known as presumed ocular histoplasmosis.

SUBJECTS AND METHODS

Thirty-one white patients in whom the clinical picture of presumed ocular histoplasmosis was present were seen in consultation. These patients were selected as the first available for testing who fulfilled the clinical criteria for presumed ocular histoplasmosis. These criteria consisted of the typically described peripheral chorioretinal atrophic scars; in some cases peripapillary scarring was also noted. A clear vitreous was present in each patient.

Additionally, a hemorrhagic or nonhemorrhagic macular lesion with disciform detachment and scarring of the sensory retina was present in at least one eve in each patient. Usually, a subretinal neovascular network was demonstrable in this area. The diagnostic examination included a complete ocular examination with pupillary dilation and indirect ophthalmoscopy, which was carried out by one or more of us. Fluorescein angiography of the macular lesion was used to demonstrate the existence of any subretinal neovascular network and to determine the need for therapy but was not a criterion for inclusion in this study. Serum was prepared for serology and fresh whole heparinized blood was collected for HLA typing. HLA typing for histocompatibility antigens was performed by using the standard National Institutes of Health microlymphocytoxicity technique. 10 At least two antisera for each specificity were used to detect the following antigens of the HLA-A and B locus:

HLA-A series—A1, A2, A3, A9, A10, A11, A28, A29, W30, W31, W32, W33, and W36:

HLA-B series—B5, B7, B8, B12, B13, B14, B18, B27, W15, W16, W17, W21, W22, W35, W37, and W40.

Splits of antigens and C locus antigens were not included in the study.

Because all patients had the typical clinical picture they were not skin-tested for histoplasmosis. This clinical picture has been accepted as diagnostic of presumed ocular histoplasmosis and it has been reported that skin testing can occasionally (7%) exacerbate a lesion in the macula.^{11–14} As many as 10 to 11% of the patients with this syndrome have demon-

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strated negative skin tests to *Histoplasma* capsulatum.^{13,14} Because of the typical clinical picture in each patient and the possibility of causing additional vision loss in already compromised maculae, skin testing was not deemed justifiable.

RESULTS

The results shown in the Table compare the incidence of HLA antigens found in our 31 white patients with presumed ocular histoplasmosis to those of two normal white populations. The frequency of HLA-B7 in our 31 patients is 54.8% as compared to 20.1% among the American Workshop control of 1,942 white patients. This increased incidence of HLA-B7 is statistically significant at $P < .005 (\chi^2 = 22.54)$ (compared with Bonferroni chi-square tables χ^2 . 995 = 14.17). The frequency in this geographical area is 20.7% and if this control figure is used P<.01 (χ^2 =13.90) (compared with Bonferroni chi-square tables which gives χ^2 . 98 =12.87). The frequencies of other antigens were calculated and compared to control frequencies from the Table. No other statistically significant correlations were found.

With respect to sex, age of onset, or serologic positivity for histoplasmosis, there is no significant statistical difference between patients who are HLA-B7 positive or negative. Homozygosity is suggested by the detection of only one antigen in a locus. Four of the patients showed only the B7 antigen present in the B locus. No significant correlation was noted in these four as far as sex or earlier onset of macular lesion.

One black patient was located with the presumed ocular histoplasmosis syndrome. Her HLA type contained (2,-) in A locus and (W17,-) in the B locus.

Serologic testing by means of a complement fixation test for histoplasmic antibodies was negative in 27 of 28 patients tested.

DISCUSSION

The clinical syndrome that has become recognized as presumed ocular histoplasmosis and its association with *H. capsulatum* has rested largely on the high but not complete correlation with a positive skin test for *H. capsulatum* in patients demonstrating this clinical syndrome^{13,14} and the correlation of the geographic distribution of this syndrome with the known positive skin test population distribution for *H. capsulatum*.^{15–17}

Although this epidemiologic evidence is convincing, many observations concerning this clinical syndrome suggest that additional factors are involved. No correlation has been made between the systemic form of the disease associated with H. capsulatum and the clinical picture of presumed ocular histoplasmosis.18-20 Treatment with amphotericin B, the drug of choice in systemic histoplasmosis, has proven unsatisfactory in the treatment of presumed ocular histoplasmosis.21 Additionally, the marked difference in racial incidence of presumed ocular histoplasmosis within the same geographic areas has previously been noted,13,14 and we were unable to locate more than one example of this clinical syndrome among the black population in this geographic area. Since blacks represent a significant portion of the population in the endemic area for H. capsulatum and since the skin test reactivity is the same in the white and black population, additional factors do appear to be involved. An inherited immunologic predisposition to alter the individual's response to the H. capsulatum organism would help explain some of these incon-

The results of histocompatibility loci typing shown in the Table substantiate a significant statistical correlation of this clinical picture with HLA-B7. The expected frequency of the histocompatibility types shown in the Table and the

TABLE HLA ANTIGEN FREQUENCY IN VARIOUS WHITE POPULATIONS

-					W40	14.3 18.9 12.9
-					W37	3.9 3.7 3.2
					W35	16.9 15.2 6.4
***************************************	W36		1.8 0 0		W22	6.4 9.1 0
	W33		2.7 0		W21	6.0 3.7 19.3
A. S. Company	W32		8.3 7.9 3.2		W17	8.9 8.5 12.9
	W31		4.9 4.4 6.4		W16	7.5 7.3 3.2
***************************************	W30		6.5 3.2 3.2		W15	11.5 12.2 9.7
***************************************	A29		7.0 5.5 12.9		B27	9.5 10.9 0
***************************************	A28	sr	9.8 8.7.4	18	B18	9.7 4.3 0
	A10 A11	A Locus	12.9 15.8 6.4	B Locus	B14	9.4 7.9 9.7
,	A10		15.7 10.9 0		B13	5.9 4.9 12.9
	A9		19.8 23.2 29.0		B12	22.9 30.5 22.6
***************************************	A3		22.6 24.4 35.5		B8	19.0 17.7 6.4
***************************************	A2		43.4 48.3 54.8		B7	20.1 20.7 54.8
	A1		26.9 28.7 22.6		B5	11.5 10.4 6.4
***************************************	No.		1942 164 31		No.	1942 164 31
тери пара вистемня от перед от настей веренения веренения веренения передовательного передовательного веренени	Antigens		American Workshop* Local Control Ocular histoplasmosis			American Workshop Local Control Ocular histoplasmosis

*HLA Workshop of the Americas. Pre-Workshop Publication, Second Cycle, New Orleans, Louisiana, April 26, 1976, M. R. Mickey (ed.) Health Sciences Computing Facility, Department of Biomathematics, University of California at Los Angeles, 1976.

statistical evaluation of these frequencies show that HLA-B7 is higher at a significant level of P<.005 among this group of 31 white patients than would be expected on a random basis, when compared with the national population, and at P<.01 when compared to the much smaller local control population.

According to recent data, additional histocompatibility loci apparently exist besides the A and B loci and these have not been tested. ²² HLA-B7 may be linked to a locus or region that determines an immunologic predisposition, influencing granuloma formation and the body's way of handling this immunologic situation. This may influence the immunologic response and lead to the development of this clinical ocular picture.

More patients should be tested to better define the meaning of this correlation with HLA-B7. It would be interesting to see what histocompatibility antigen frequency could be found in a group of patients satisfying these clinical criteria and living in areas nonendemic for *H. capsulatum*.

We are reporting our results on this initial study in the hope that it will encourage others to consider histocompatibility antigens and specifically HLA-B7 and its role when future attempts are made to understand the uncertain etiologies and mechanisms that produce the clinical entity known as presumed ocular histoplasmosis.

SUMMARY

Thirty-one white patients who fulfilled the clinical criteria of the syndrome recognized as presumed ocular histoplasmosis were typed for common histocompatibility antigens. These clinical criteria included the presence of multiple peripheral punched out choroidal atrophic scars, compatible macular disciform lesion in at least one eye, and clear vitreous. Seventeen out of 31 patients were found to have

HLA-B7, which is statistically significant at the P<.005 level when compared to a normal population. More patients should be tested to establish this correlation more firmly. Though this is statistically significant, other factors must be involved as there still remain many patients who fulfill the clinical criteria but do not demonstrate a common histocompatibility antigen.

ACKNOWLEDGEMENTS

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THE PREVALENCE OF HLA-B7 IN PRESUMED OCULAR HISTOPLASMOSIS

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The presumed ocular histoplasmosis syndrome is defined as a clinical triad of disciform disease of the macula, peripapillary atrophy, and peripheral chorioretinal scarring. A minority of patients with evidence of past systemic histoplasmosis have evidence of ocular involvement. In the Wakersville, Maryland, epidemiologic study, 59% of the population were found to have positive histoplasmin skin tests and, among those with positive skin tests, 4.4% were noted to have peripheral scarring typical of histoplasmosis. 1,2 Only 0.2% of those with positive skin tests had macular disciform scars. 1,2 This study reports the results of HLA typing done in a group of patients with the presumed ocular histoplasmosis syndrome.

SUBJECTS AND METHODS

We reviewed the files of the Retina Section at the Medical College of Wisconsin and The Milwaukee County Medical Complex for patients with presumed ocular histoplasmosis syndrome. The criteria for including patients in this study were: (1) at least two of the three clinical features of presumed ocular histoplasmosis syndrome; and (2) a documented actively bleeding lesion in the posterior pole at some time. Eighteen white patients met these criteria and were typed for histo-

compatability antigens according to the National Institutes of Health lymphocyte microcytotoxicity technique.³

The antigen frequency of the control population is based on the typing of 92 unrelated white individuals employed at the Milwaukee County Medical Complex.

Statistical treatment of the results was performed using the chi-square test incorporating the Yeats correction factor as described by Svejgaard and co-workers⁴ for HLA and disease studies. The P value associated with each chi-square determination was multiplied by 30, the number of antigens studied.

RESULTS

We have summarized the clinical features of the patients and their HLA types at the A and B loci (Table). Of the 18 patients studied, 14 were found to have HLA-B7. Compared with the frequency of HLA-B7 in the control population (20%), the association of the presumed ocular histoplasmosis syndrome patients and HLA-B7 is considered statistically highly significant (P<.0003). There is no statistical significance between presumed ocular histoplasmosis syndrome and any of the other HLA antigens.

The strength of an association is estimated by the relative risk.⁶ Relative risk is defined as:

(% antigen pos. pts.) (% antigen neg. controls) (% antigen neg. pts.) (% antigen pos. controls) The relative risk for the observed association between presumed ocular histoplasmosis syndrome and HLA-B7 is 11.8.

DISCUSSION

Seventy-eight percent of the patients in this study having the clinical diagnosis

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TABLE
THE CLINICAL FEATURES OF PATIENTS AND THEIR HLA TYPES
AT THE A AND B LOCI*

Patient No.	Disciform Scar	Peripapillary Atrophy		Antigens		
			Peripheral Scars	Locus A	Locus E	
1	+	+	+	1,2	7,8	
2	+	+	+	1,3	7,8	
3	+	+	+	3,W32	7,W15	
4	Name .	+	+	W23,29	7,12	
5	+	+	ATT	1,2	7,27	
6	***	+	+	3,W33	7,12	
7	+	+	+	2,W24	7,27	
8	+	+	+	1,3	7,W37	
9	+	+	+	2,11	7,W21	
10	+	_	+	1,3	7,8	
11	+	+	+	2,3	7,-	
12	+	+	+	1,-	27,W35	
13	+	+	+	1,2	7,8	
14	+	_	+	1,	8,	
15	atre	+	+	28,W26	8,W16	
16	+	+	+	3,W23	12,W35	
17		+	+	2,-	$7,\!27$	
18	+	+	+	1,3	7,13	

^{*+}indicates presence of a particular feature; -, absence.

of presumed ocular histoplasmosis syndrome and a documented active posterior pole lesion were found to have HLA-B7. The HLA system is considered to have a major role in determining susceptibility to a diverse group of diseases, including ankylosing spondylitis,7 dermatitis herpetiformis,8 primary open-angle glaucoma,9 anterior uveitis,10 and recurrent corneal herpes infections. 11 These types of data suggest that patients develop some diseases through an inherent predisposition. Many of these disease associations are considered to be caused by the effect of the immune response or immune suppression genes determining altered individual responsiveness to foreign antigens. There is evidence of linkage between HLA antigens and immune response genes. Therefore, rather than a direct correlation with genes found at HLA loci, it may be the immune response genes that are being identified in association with certain disease states.7

In the development of an active lesion

in ocular histoplasmosis, there are probably two separate and distinct stages. In the first stage, there is an infection of the choroid with *Histoplasma capsulatum* that leaves scars in the fundus. The second stage is the development of subretinal neovascularization and an actively bleeding lesion. This has been postulated on epidemiologic grounds to occur 10 to 30 years after the first stage.¹²

The occurrence of subretinal neovascularization is now recognized to occur around previously established scars^{14–16} when it occurs in the fellow eye of patients with a disciform lesion. This change has been postulated to be caused by either a central decompensation of a longstanding atrophic lesion^{14,17} or some active immunologic phenomenon.^{18–20}

The increased susceptibility of these patients implied by our data might be to the initial event of infection of the choroid by the *H. capsulatum* organism, although in the experimental model the number of choroidal lesions is propor-

tional simply to the infective dose rather than any other known factor. ¹³ That the point of susceptibility is an increased risk of late decompensation mediated at least in part by immunologic mechanisms is an attractive hypothesis. However, our data cannot clearly distinguish whether the predisposition is for the initial eye involvement or for later decompensation; further studies in patients with other forms of the disease may be helpful.

The percentage of HLA-B7 in this group of patients rivals the incidence of positive histoplasmin skin tests in patients with the clinical syndrome. HLA typing may be useful in further population studies of presumed ocular histoplasmosis syndrome, as well as in diagnosing difficult clinical presentations of posterior pole lesions.

SUMMARY

Eighteen patients with the presumed ocular histoplasmosis syndrome were typed for HLA histocompatability antigens and 78% were found to have the B7 antigen. This data implies an underlying immunogenic predisposition to the development of the clinical eye findings and active posterior pole lesions in the presumed ocular histoplasmosis syndrome. It is likely that some feature of the immune system of these patients renders them more susceptible either to the initial infection or to a later activation of posterior pole lesions mediated through immunologic mechanisms.

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HYPERTENSIVE REACTIONS TO PHENYLEPHRINE EYEDROPS IN PATIENTS WITH SYMPATHETIC DENERVATION

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Ten percent phenylephrine eyedrops (5 to 7.5 mg per drop)1 have been used topically in various eve operations, either pre- or intraoperatively. Undesirable systemic effects are known to be rare.^{2,3} Previous studies revealed three cases of hypertension⁴ and one of subarachnoid hemorrhage caused by severe hypertension.⁵ These episodes were mainly ascribed to overdose of phenylephrine eyedrops. We investigated some hypertensive reactions to ordinary doses of phenylephrine eyedrops to determine whether these might be caused by supersensitive reactions in patients whose sympathetic nervous systems are partially denervated, as in insulin-dependent diabetic patients, or in patients who had been taking reserpine or guanethidine.

SUBJECTS AND METHODS

The study was conducted on 298 patients: 271 underwent ocular surgery, and 27 underwent extraocular surgery under general anesthesia or regional block with anesthesia standby. An anesthesiologist visited all patients the day before surgery and reviewed their charts, obtained additional history, and explained the planned anesthesia procedure. Preanesthetic med-

ications were ordered for the patients scheduled for general anesthesia, but no preliminary sedative was administered to the patients for anesthesia standby. When the patient arrived in the operating suite, laboratory data and time and dosage of administered eyedrops and of preanesthetic medications were recorded. Most patients who received regional block with anesthesia standby were given neuroleptanalgesia (intravenous droperidol and fentanyl).

The ophthalmologist ordered mydriatics as necessary. Beginning two hours before the operation, one or two drops of 10% phenylephrine, alone or combined with 1% atropine or 1% cyclopentolate or both, were administered every 15 minutes for a total of three or four doses. On admission of the patient to the ward, the nurse measured the blood pressure, which was considered the patient's resting level, before medication. The anesthesiologist measured the preoperative blood pressure just before the administration of anesthetics. The time that elapsed from the administration of the last dose of eyedrops to the measurement of the preoperative blood pressure was termed "interval minutes.'

The patients were divided into three groups, and then into two subgroups (A and B).

Group 1 (176 and 54 patients in subgroups A and B, respectively) had no history of insulin-dependent diabetes or of prior treatment with reserpine or guanethidine.

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Group 2 (14 and 27 patients in subgroups A and B, respectively) comprised the insulin-dependent diabetic patients.

Group 3 (12 and 15 patients in subgroups A and B, respectively) comprised the hypertensive patients who had been taking reserpine or guanethidine.

Patients in subgroup A of each main group (202 patients) underwent ocular surgery preceded by topical administration of phenylephrine eyedrops. Patients in subgroup B of each main group did not receive phenylephrine eyedrops. Extraocular surgery was performed in 18 of 27 patients in Group 2B, and nine of 15 patients in Group 3B. The data on these 27 patients were collected from anesthesia records selected at random from recent files. Preoperative blood pressure as compared to admission blood pressure was determined in each patient.

RESULTS

Preoperative blood pressure, as compared to admission blood pressure, was not statistically significant in all B subgroups (the 96 patients who did not receive preoperative phenylephrine eyedrops) (Table 1). Also, there was no significant difference in Group 1A (the 176 patients who received preoperative

phenylephrine eyedrops but were neither insulin-dependent diabetics nor taking sympatholytic drugs). However, after administration of phenylephrine eyedrops, both systolic and diastolic pressures insignificantly in creased insulindependent diabetic patients (Group 2A) and in patients who had been taking reserpine or guanethidine (Group 3A). The mean increases in systolic and diastolic pressures were 34.2 and 16.7 mm Hg, respectively, in Group 2A; and 30.0 and 13.0 mm Hg, respectively, in Group 3A. Changes in both systolic and diastolic pressures were statistically significant (P<.01) in Groups 2A and 3A. Occasionally, there were alarming rises in pressure.

The hypertensive reaction to phenylephrine eyedrops was more reliably predictable the longer the duration of the diabetes, and the older the patient who had been taking sympatholytic drugs (Tables 2 and 3). Mean duration of diabetes in Group 2A was 13.8 years. Mean age of Group 3A was 72.3 years, as compared to 61.5 in Group 3B.

DISCUSSION

Phenylephrine increases both systolic and diastolic pressures by vasoconstric-

TABLE 1

Changes in preoperative blood pressure as compared to admission blood pressure in each of six subgroups

Group	Phenylephrine Eyedrops	No. of Patients	Age (mean)	Interval Minutes* (mean)	Systolic Gradient† (mean ± SD)	Diastolic Gradient‡ (mean ± SD)	Anesthesia General Regional	Comments
1A	Yes	176	58.1	70	0.3 ± 20.0	-0.2 ± 14.7	113/63	
IB	No	54	57.5		-4.6 ± 12.4	-1.6 ± 12.4	29/25	
2A	Yes	14	51.6	71	34.2 ± 27.4 §	16.7 ± 18.3§	6/8	Insulin-dependent diabetic patients
2B	No	27	52.9		0.0 ± 20.2	5.0 ± 16.1	19/8	
3A	Yes	12	72.3	85	30.0 ± 30.2 §	13.0 ± 13.2§	7/5	Patients receiving reserpine or guanethi-
3B	No	15	61.5		2.8 ± 16.0	-0.4 ± 13.6	11/4	dine

^{*}Interval minutes: the elapsed time from the last dose of eyedrops administered to the time when the preoperative blood pressure was measured.

1Systolic gradient: preoperative systolic pressure-systolic pressure on admission.

Diastolic gradient: preoperative diastolic pressure-diastolic pressure on admission

TABLE 2

Changes in blood pressure following administration of phenylephrine eyedrops in insulin-dependent diabetic patients

Age (yrs)	Interval Minutes	Admission Blood Pressure / mm Hg /	Preoperative Blood Pressure / mm Hg /	Duration of Diabetes (yrs)	Comments†
*52	45	160/80	200/110	27	Lente 32 units/day Neuropathy
*53	75	120/70	210/100	28	Lente 32 units/day Neuropathy
51	95	146/86	200/120	15	NPH 35 units/day
59	25	150/72	170/110	20	NPH 50 units/day
61	60	130/80	150/70	5	NPH 40 units/day
70	55	130/88	130/80	2	NPH 15 units/day
24	85	140/80	180/95	16	NPH 18 + Reg insulin 8 units/day
21	120	100/60	110/80	3	NPH 40 + Reg insulin 18 units/day
64	145	140/70	165/90	20	NPH 40 units/day
51	70	170/100	200/110	20	NPH 30 units/day
$\tilde{73}$	80	170/88	170/75	2	Lente 10 units/day
64	45	112/40	160/75	15	Lente 25 units/day Neuropathy
59	35	200/100	210/90	10	NPH 25 units/day Retinopathy
24	55	110/80	190/115	11	Lente 0 units/day Neuropathy

^{*}Same patient.

tion, without altering chronotropic and inotropic actions on the heart. The pressor effect lasts about 20 minutes after intravenous injection, and as long as 50 minutes after subcutaneous administration. Doses estimated to produce similar pressor responses are 0.8 mg intravenously, 5 mg subcutaneously or intramuscularly, and 250 mg orally.6 The degree of hypertension depends on the free plasma phenylephrine concentration available to the vascular receptors which, in turn, is mainly determined by the balance between the rate of systemic absorption and of inactivation. The extent of systemic absorption that occurs is a function of the total dose and the route of administration. The major mechanisms responsible for inactivation are diffusion, uptake by axonal terminals, and absorption by other cells.

The effective plasma concentration of phenylephrine after topical ocular administration is not predictable. The amount subject to systemic absorption can be altered by overflow waste, dilution caused by blinking and reflex tear production, or simultaneous use of other evedrops, as atropine and cyclopentolate. Only the small fraction of the phenylephrine retained in the eye (less than one drop) and which drains into the nasal mucosa, can be systemically absorbed. Absorption through the intact conjunctiva is admittedly slow and limited in degree. If the terminating mechanisms of phenylephrine activity are intact, the usual clinical doses of topical phenylephrine eyedrops rarely cause hypertensive reactions, as demonstrated by the patients in Group 1A. This confirms the same finding reported in a previous study by Reynolds, Havener, and Henderson.⁵

A hypertensive blood level can occur with excessive doses and with the loss of the blinking reflex and suppressed lacrimal secretion during anesthesia.⁷ Excessive doses may be administered to small

fInsulin zinc suspension (Lente); isophane insulin suspension (NPH).

TABLE 3

Changes in blood pressure following administration of phenylephrine eyedrops in hypertensive patients receiving reserpine or guanethidine

Age / yrs /	Interval Minutes	Admission Blood Pressure / mm Hg/	Preoperative Blood Pressure / mm Hg /	Antihypertensive Medicine	Comments
64	110	165/90	170/110	Guanethidine	
71	100	150/75	240/100	30mg twice a day Reserpine (Salutensin) 1	
80	60	124/80	190/100	twice a day Reserpine (Ser-Ap-Es) 1	Hypokalemic
59	115	150/80	130/70	twice a day Reserpine (Regroton)	
73	60	150/85	200/100	1/day Reserpine (Ser-Ap-Es)	Phenylephrine 2 drops, 4 times a day
80	90	130/60	170/90	1/day Reserpine (Regroton)	Phenylephrine 2 drops, 4 times a day
75	80	140/80	190/90	1/day Reserpine (Ser-Ap-Es)	
77	70	180/90	180/80	1/day Reserpine (Ser-Ap-Es)	
74	55	180/80	190/110	1/day Reserpine (Ser-Ap-Es)	Stroke ten years ago
67	90	150/80	180/90	1/day Reserpine (Salutensin)	
79	100	150/84	170/100	1/day Deserpidine (Enduronyl)	
69	90	150/80	170/80	1/day Reserpine (Ser-Ap-Es) 1/day	

children and to patients who receive drops in both eyes. This type of hypertension was observed in several patients in Group 1A. We also observed both hypertension and cardiac arrhythmia in a young healthy man who received three drops of phenylephrine intraoperatively. Similar hypertensive reactions were reported by Solosko and Smith.⁴

In contrast to Group 1A, similar doses

of phenylephrine eyedrops significantly increased both systolic and diastolic pressures in the insulin-dependent diabetic patients (Group 2A), and in the patients who had been taking reserpine or guanethidine (Group 3A). If a similar amount of drug is absorbed systemically in all patients, the hypertensive reactions in Groups 2A and 3A can only be explained on the basis of unusual sensitivity to

phenylephrine or impaired terminating mechanisms of the drug.

Supersensitivity to catecholamines in sympathetically denervated patients has been well documented. Because the overall uptake of catecholamine by adrenergic neurons is reduced in these patients, more free agonist is available to react with tissue receptors. Sympathetic denervation is common to the patients in Groups 2A and 3A: autosympathectomy by autonomic neuropathy in the former, and pharmacologic sympathectomy in the latter.

Many diabetic patients undergo ocular surgery for retinopathy and for removal of cataracts. Neuropathy and nephropathy are often associated complications that could share a common etiology.10 Most diabetic patients with these complications have long histories of insulin dependence. According to the report by Sharpey-Schafer and Taylor,11 31 of 39 diabetic patients with neuropathy demonstrated autonomic dysfunction. Fekete, Rub, and Bogdan¹² reported that 11 of 26 young diabetic patients showed evidence of cardiac autonomic neuropathy. Neubauer and Gunderson¹³ noted a relationship between cardiac autonomic neuropathy and the duration of diabetes. These reports demonstrate the high incidence of autonomic neuropathy, especially among long-standing insulin-dependent diabetic patients. The mean duration of the disease in our patients in Group 2A was 13.8 years. Lloyd-Mastyn and Watkins¹⁴ described a diabetic patient with proven autonomic neuropathy, whose systolic pressure increased by 54 mm Hg after intravenous administration of 0.05 mg of phenylephrine; this equals 1/100 of one drop of 10% phenylephrine.

Reserpine and guanethidine are sympatholytic agents used for control of essential hypertension. Prolonged administration of these drugs induces super-

sensitivity of effector cells similar to that produced by sympathetic postganglionic denervation. Hypertensive crises may follow the use of remedies for the common cold which contain sympathomimetics.6 Also, the use of local anesthetics containing epinephrine for regional block must be used cautiously. Ten milliliters of a local anesthetic solution with 1:200,000 epinephrine contains 0.05 mg of epinephrine. The supersensitivity persists for several days after the discontinuance of sympatholytic drugs. 6,15 We observed long-lasting supersensitivity in one patient (the first patient in Table 3). In spite of discontinuance of guanethidine five days before surgery, the blood pressure rose from 165/92 mm Hg to 210/120 mm Hg soon after the administration of phenylephrine eyedrops. The pressure then declined to 170/110 mm Hg without treatment in the following 100 minutes, while the patient was still in the operating room.

The supersensitivity reaction can be manifested regionally in the iris. Prolonged and profound mydriasis is undesirable for certain operations and uncomfortable for the patient. Cooper¹⁶ reported prolonged mydriasis (up to ten hours) in one patient who had been receiving oral guanethidine. The mydriasis was not reversed by miotics. The sensitive patient often does not require a full dose of phenylephrine to obtain adequate mydriasis. Thus, both systemic and regional supersensitivity reactions can occur.

Most of our supersensitivity hypertensive episodes were readily controlled without further incident by administration of general anesthesia with halothane and by neuroleptanalgesia accompanying regional block with anesthesia standby. Halothane decreases blood pressure by myocardial depression and vasodilation.¹⁷ During anesthesia with halothane,

the cardiac arrhythmogenic threshold to epinephrine decreases, but this is not the case with phenylephrine. 18 Sprague. Yang, and Ngai¹⁹ reported that phenylephrine-induced aortic contraction was inhibited by halothane in a dosedependent manner. The protective action of neuroleptics against catecholamineinduced vasoconstriction has been demonstrated, but seems to be nonspecific.20

These supersensitivity reactions are avoidable. The dosage of phenylephrine should be carefully titrated in patients with insulin-dependent diabetes, or another mydriatic that does not have alpha adrenergic activity should be administered. The same is true for patients receiving reserpine or guanethidine if these drugs cannot be withdrawn safely for more than ten days before surgery.

SUMMARY

We studied the effects of topical phenylephrine eyedrops on systemic blood pressure in 298 patients about to undergo ocular surgical procedures by comparing their blood pressure on admission to the hospital with that measured immediately before surgery. The patients were divided into three groups. Group 1 consisted of 230 patients who had neither history of insulin-dependent diabetes nor prior treatment with reserpine guanethidine. Group 2 included 41 insulin-dependent diabetic patients. Group 3 contained 27 hypertensive patients who had been taking reserpine or guanethidine. Patients in each group were divided into two subgroups (A and B). The 202 patients in the three A subgroups received preoperative phenylephrine evedrops, whereas the 96 patients in the three B subgroups did not.

All three B subgroups and Group 1A (176 patients) did not show significant increases in blood pressure. There was a statistically significant increase in both systolic and diastolic pressures in Group 2A (14 patients) and in Group 3A (12 patients). From this study, we concluded that administration of preoperative phenylephrine eyedrops can be hazardous in patients with long-standing insulin-dependent diabetes or in hypertensive patients receiving reserpine or guanethidine.

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OPHTHALMIC MINIATURE

"Here, waiter," shouted the stranger, ringing the bell with tremendous violence, "glasses round, brandy and water, hot and strong, and sweet, and plenty eye damaged, sir? Waiter; raw beef-steak for the gentleman's eye, nothing like raw beef-steak for a bruise, sir; cold lamp-post very good, but lamp-post inconvenient damned odd standing in the open street half an hour, with your eye against a lamp-post eh, very good ha! ha!"

Charles Dickens, Pickwick Papers

NOTES, CASES AND INSTRUMENTS

A SIMPLE DEVICE TO IMPROVE BLINKING

MARK S. JENKINS, M.D., PAUL G. REHKOPF, AND STUART I. BROWN, M.D. Pittsburgh, Pennsulvania

Incomplete or infrequent blinking has been shown to cause drying of the precorneal tear film and desiccation and erosion of superficial corneal epithelium.^{1,2} Infrequent or partial blinking is also a major cause of hard contact lens intolerance.³ In our experience, simply telling the patient of the need for proper blinking consistently meets with failure. We report on a simple gadget that has been successful in improving alterations in blinking and their resultant epithelial erosions.

SUBJECTS AND METHODS

In order to promote blinking in patients with normal orbicularis function, but infrequent or incomplete blinking, a blink beeper was devised; it emits an audible tone through a small beeper contained within the device or transmitted from the device through an earphone placed in the user's ear. This device is approximately the size of a package of filter-tip cigarettes (4 inches \times 2½ inches \times 7/8 inch); it can thus be concealed in a pocket or under the user's clothing (Figure). The audible tone occurs every ten seconds. The volume of the tone is adjustable with a control on the outside of the device that the user may adjust himself. The power to



Figure (Jenkins, Rehkopf, and Brown). The blink beeper.

operate the device is derived from an AAA 1.5-volt alkaline battery that lasts approximately a month in continuous operation. The case is made of high-impact plastic with all electronic components mounted on a printed circuit board, thus making this device relatively shock-resistant.

CASE REPORTS

Case 1—This 15-year-old boy had a history of congenital glaucoma, which after numerous operations, resulted in enucleation of his blind, painful right eye. The left eye had five surgical procedures performed to reduce the intraocular pressure (IOP) and two years ago a cataract extraction followed by a cyclocryothermy. In April 1976, the patient was examined here and was found to have good light projection. The eye was grossly buphthalmic with a corneal diameter of 15 mm; the cornea was diffusely edematous and the IOP was 17 mm Hg. Shortly thereafter, he underwent a 7.5-mm penetrating keratoplasty. Postoperatively, the graft remained transparent, but developed numerous superficial punctate erosions that eventually coalesced to form a large central erosion soon after the patient was allowed to keep his eyelids open. These erosions healed with patching but recurred shortly after the eves were allowed to remain open. The erosions were thought to be caused by the patient's infrequent and incomplete blinking, which we believed was associated with his many years of blindness. The patient was repeatedly told of the need to blink, but continued to have infrequent and partial blinking. A soft contact lens was fitted, which together with hourly topically applied artificial tears, maintained the integrity of the corneal epithelial surface. Eight months postoperatively, the patient developed a corneal stromal infiltrate within a small epithelial erosion from which mannitol-positive Staphylococ-

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cus was cultured. Intensive antibiotic treatment resulted in eventual healing. One year postoperatively, the patient returned with a circumferential liquefaction of the periphery of the corneal graft; the graft was heavily infiltrated and showed Pseudomonas aeruginosa on both smears and cultures. A 7.5-mm penetrating keratoplasty was performed and, postoperatively, the graft remained transparent. When the eye was allowed to open, superficial punctate epithelial erosions were once more observed. To avoid a soft contact lens and the possible complication of infection, a blink beeper was given to the patient. He was instructed that each time he heard the beep, he was to blink and feel his upper evelid touch the lower eyelid. The beeper was removed for sleep only. No epithelial erosions were seen and, after two months, the beeper was taken from the patient for a one-week trial. His blinking continued to be complete and frequent. There have been no epithelial erosions for the past six months.

Case 2—This 56-year-old man was admitted to the hospital here with a large central corneal perforation secondary to herpes simplex keratitis. The perforation was treated with a 7.5-mm penetrating corneal transplant. Postoperatively, there were extensive peripheral and central anterior synechiae. The lens became increasingly intumescent, which further decreased the depth of the anterior chamber, and the IOP increased. A second corneal transplant combined with a cataract extraction was performed. Postoperatively, the chamber remained deep with the pressure under control. The host epithelium did not completely heal over the graft; this resulted in a central epithelial defect between 5 and 6 mm in diameter, which did not show any sign of healing over a five-month period despite repeated debridement and various soft contact lenses. Pressure patching was attempted and discontinued when it was observed that the eyelids were still open under the apparently firm dressings. The cause of the slow healing epithelium was considered a complication of grafting a cornea with herpes simplex keratitis that was aggravated by the patient's widened palpebral fissures and infrequent blinking (one to two blinks per minute). The patient was given the blink beeper five months postoperatively. One week later the epithelial defect was 3 to 4 mm in diameter, and after two weeks it was healed. The beeper was taken from the patient after six weeks and the epithelium has remained healed for the past three months.

Case 3—This 67-year-old woman had a history of bilateral intracapsular cataract extractions when she was 55 years old. Within the first year postoperatively she developed bullous keratopathy in both eyes. In September 1976, she underwent a 7.5-mm penetrating keratoplasty in the right eye. Postoperatively, the graft remained transparent but developed punctate epithelial erosions; the erosions coalesced to form a 4 × 5-mm epithelial defect when the eyelids were allowed to remain open. This patient also had widened palpebral fissures and a blink rate

of approximately two blinks per minute, despite frequent coaxing to blink.

The epithelial defect repeatedly healed with patching, but would erode shortly after allowing the eyelids to remain open in spite of the use of a soft contact lens and hourly artificial tears. Four months postoperatively, a Henderson procedure was performed; this reduced the width of the patient's palpebral fissure and the epithelial defect decreased in size, but never to less than 2 × 3 mm unless patched. Six months postoperatively, the patient was given a blink beeper. Two weeks later her corneal epithelial defect had healed. After two months of using the blink beeper, a one-week trial period was attempted to see if the patient would continue to blink without the device. No erosions occurred and the device was taken from the patient. Thereafter, only occasional punctate epithelial erosions have been observed during a three-month follow-up peri-

SUMMARY

A blink beeper was devised to remind patients with incomplete or infrequent blinking to blink completely and at normal intervals; the beeper worked by transmitting an audible tone every ten seconds through an earphone. In three postkeratoplasty patients, this simplistic approach not only resulted in a complete blink at regular intervals, but seemed to condition this response to continue when the blink beeper was taken away; this resulted in healing of large chronic epithelial defects. The blink beeper has also proved valuable in patients with either punctate epithelial erosions or those who wore hard contact lenses unsuccessfully because of infrequent or incomplete blinking.

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ZINC DEFICIENCY AND VISUAL IMPAIRMENT?

Zinc as mentioned in the Ebers papyrus of 1550 B.C.1 had always been thought to be an inert substance until Raulin² noted that Aspergillus niger would not grow in its absence.

Keilin and Mann³ found zinc to be an essential part of the enzyme carbonic anhydrase, the first metalloenzyme to be discovered. Subsequently, zinc has been shown to be a component of several enzyme systems, which include alchol dehydrogenase, alkaline phosphatase, and enzyme systems involving nucleic acids and protein and carbohydrate metabolism.4

Zinc is a structural and functional component of the digestive enzyme carboxypeptidase and participates directly in the catalytic activity of this enzyme.⁵ Zinc is also found in the insulin molecule. These observations provide sufficient reason to believe that humans need zinc.

The highest concentration of zinc in the human tissues occurs in the prostate, spermatozoa, and parts of the eye.6 Meat,

liver, eggs, milk, whole grains, and seafoods, especially oysters, are good sources of zinc, whereas fruits, leafy vegetables, bread, fat, and sugar are poor sources of it.

Zinc is necessary to maintain the normal concentrations of vitamin A in the plasma and is involved in the mobilization of vitamin A from the liver.⁷

An international symposium on zinc focused attention on the clinical aspects of zinc metabolism.8 Zinc deficiency in animals and plants is marked by decreased protein synthesis associated with depressed activity of both RNA and DNA polymerases.9

In human beings, zinc deficiency may be teratogenic. 10,11

There has been an unresolved conflict in previous studies concerning the role of zinc in the healing of thermal and excised wounds.12,13

Recently, Moynahan^{14,15} established the crucial role of zinc deficiency in acrodermatitis enteropathica, a serious illness that starts early in infancy after weaning.16,17 The pathogenesis of the disease

is unknown. As the name implies, there are prominent gastrointestinal symptoms, especially diarrhea. The skin of the hands, feet, perioral region, and orifices shows symmetrical demarcated vesicles bullae and erythema with crusting. Alopecia occurs, and the eyes usually show photophobia, blepharitis, and conjunctivitis. Linear subepithelial corneal opacities and radiating lines may be the results of the disease. 18–20

Many other researchers throughout the world have confirmed this outstanding observation, that is, patients with low plasma zinc levels respond rapidly to zinc therapy.^{21–24}

Until this discovery, the only effective therapy for this otherwise fatal disease involved long-term treatment with diiodohydroxyquin, and the mechanism by which it works has not been determined. In rare instances, survival through this therapy was accompanied by optic atrophy. However, one cannot eliminate the possibility that the nerve involvement is due to the underlying disease, namely, the zinc deficiency, and not to the drug.

Moynahan's discovery has implications far beyond the small group of seriously ill patients with acrodermatitis enteropathica, who will be the immediate beneficiaries. His discovery should serve as a powerful stimulus to all interested in zinc metabolism to look for other manifestations of deficiency, either natural or induced, in both children and adults.

Diiodohydroxyquin is a potent metal binding agent. Perhaps its chelation of dietary zinc aids zinc absorption through the intestinal barrier and proceeds to carry it to the tissues so that the drug then acts simply as a zinc carrier. Possibly, the diiodohydroxyquin binds a substance or ligand in the intestinal tract that competes for the zinc and thus prevents the zinc from binding at these sites and frees it for absorption.

A deficiency of zinc may play a role in the pathogenesis of certain toxic and nutritional optic neuropathies. Buyske, Sterling, and Peets²⁵ first hypothesized that the toxic effects of ethambutol, particularly on the optic nerve, might be related to the in vivo chelating action of this product on metallic ions. Three years earlier Vogel and Kaiser²⁶ demonstrated that the in vivo administration of ethambutol produced a reversible bleaching of the tapetum lucidum, a structure containing a large zinc concentration. Figueroa²⁷ reported that this loss of pigmentation is accompanied by a decrease of the zinc content of the organ. Saraux, Bechetoille, Nou, and Courtois²⁸ showed that the serum zinc level determined by atomic absorptive spectrometry was greatly diminished in three cases of ethambutolinduced optic neuritis, one case of disulfiram-induced optic neuritis, and seven cases of optic neuritis due to alcholism and tobacco intoxication.

A deficiency of zinc or other metallic trace elements might play a role in the pathogenetic development of certain toxic and nutritional optic neuropathies. Many products, drugs, industrial pollutants, and food products that reduce the serum level of numerous metallic cations, particularly zinc, also show a certain toxicity for the optic nerve. The absence of a certain number of nutrients can lead to optic neuritis. All of these toxic neuropathies have a similar clinical picture. Their onset is marked by dyschromatopsia of the red/green axis, and the neuritis is usually retrobulbar in the early stages.

A number of drugs besides ethambutol may also reduce serum zinc level: disulfiram, oxyquinolines, diiodohydroxyquin, penicillamine, DL-penicillamine, isoniazid, and certain monoamine oxidase inhibitors such as iproniazid, nialamide, isocarboxazid. A diminished serum zinc

level was demonstrated by El-Gazzar, El-Sadik, and Hussein²⁹ in individuals exposed to carbon disulfide in a recent accident in an Egyptian synthetic textile plant.

Chronic alcholism with or without cirrhosis is accompanied by reduction of serum zinc level.

The tannins in low quality red wines form stable chelates with the metal cations in the digestive tract; they are eliminated with the feces and thus cause absorption insufficiency.

The phytates in cereals and soybeans can also cause absorption insufficiency by precipitation of the metals in the digestive tract.

Diminished serum zinc levels have been observed in malabsorption syndromes, as in gastrectomized patients, and in starvation syndromes, particularly in kwaschiokor, a deficiency disease. In these types of deficiency diseases, one cannot assume simply a deficiency of the metal trace elements because many patients with depressed serum zinc levels do not develop optic neuropathy and certain chelating drugs have never been incriminated in the etiology of toxic optic neuropathy.

Galin³⁰ determined that the ocular tissues, in man as well as in animals, contain high concentrations of zinc. The optic nerve also contains large quantities, approximately 120 mg/kg of body weight. The choroid and the retina, which is reported to have 463 mg/kg of body weight of dried tissue, have two of the highest concentrations of zinc of all the specialized tissues of the body. The retinal reductase, which is involved in the reciprocal conversion of vitamin A into retinene, is probably a zinc enzyme. Vitamin A is transported from its storage site in the liver to its target site by plasma in a form bound to a specific protein that depends on zinc for its synthesis.

Zinc is found in high concentration in uveal and retinal tissue. O'Rourke, Durrani, Benson, Bronzina, and Miller³¹ believe that this is an important trace element in uveal and retinal metabolism. In several species, these tissues contain the body's highest concentrations of zinc. and several zinc-containing enzymes such as carbonic anhydrase and lactic and alcholic dehydrogenases important to uveal or retinal function are present at high levels in these layers. Tu, Blackwell, and Lee³² and Gibbs and Walshe³³ described patients with optic neuritis who received DL-penicillamine and had prompt reversal of the lesion with pyridoxine therapy. Klingberg, Prasad, and Oberleas³⁴ failed to reverse the optic neuropathy associated with penicillamine therapy by giving large doses of pyridoxine; however, the eye in their case did not respond to subsequent zinc therapy. Possibly the therapy was instituted after irreversible changes had occurred. They attributed the low zinc levels in the body to the chelation effect by penicillamine. which depleted the body of its zinc content.

The optic neuropathy attributed to the use of diiodohydroxyquin in acrodermatitis enteropathica may be related to this chelating effect on zinc, for the gastrointestinal and skin signs associated with this disease can be reversed by the use of zinc. The optic atrophy in acrodermatitis treated with diiodohydroxyquin may be a zinc deficiency sign rather than a specific sign of toxicity of the drug. The drug penetrates poorly from the intestinal tract. More likely, it binds a ligand in the intestinal tract and thus frees the zinc, as previously mentioned.

Other forms of optic neuropathy, whether previously diagnosed or not, might benefit from zinc administration. The tissue and zinc levels of such patients should be analyzed before therapy.

Although photophobia is often mentioned in discussing acrodermatitis enteropathica, little or no attention has been paid to the striking disturbance of visual behavior in acrodermatitis enteropathica. Although many published photographs show the gaze aversion characteristic of the disorder, the avoidance of the eyeto-eye contact is often an unappreciated feature. The child finds a central cone vision distressing and therefore relies as much as possible on peripheral vision, which is probably dependent on rods. Movnahan³⁵ speculated that the autistic child who displays similar aberrant visual behavior may be in the same visual predicament. The sensitivity of the cone caused by lack of zinc is further corroborated by Michaelsson's²² patients whose color vision, which had been lost somewhat before diiodohydroxyquin associated optic atrophy, returned promptly when the patient was given zinc.22 The eve is rich in zinc, expecially the retina, but it is not clear why the cones seem to be more susceptible to zinc deficiency than the rods.

Four cases of reduced serum zinc have been reported in hereditary optic atrophy in early childhood³⁶ and in a beer drinking patient with a Billroth resection with chronic zinc deficiency.³⁷

These compelling observations suggest we should carefully examine our patients with optic neuropathy and perhaps retinal disorders of unknown etiology for the possibilities of zinc deficiency.

IRVING H. LEOPOLD

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OBITUARIES

Sir Stewart Duke-Elder 1898–1978

With the death of Sir Stewart Duke-Elder on March 27, 1978, 26 days before his 80th birthday, the world of ophthalmology lost its most famous and most distinguished figure. By his unprecedented contributions to ophthalmology as a scientist, surgeon-oculist, leader, innovator, and, above all, as an author of rare talent, he is acknowledged as one of the greatest ophthalmologists the world has known.

William Stewart Duke-Elder was born in the village of Tealing near Dundee in Scotland on April 22, 1898, and was the second of three sons born to Neil Elder, a minister in the Free Church of Scotland, and Isabella (née Duke). His early upbringing was therefore in an atmosphere of disciplined authority and limited means, restrictions which no doubt played some part in directing his youthful energies towards study and scholastic achievement rather than to idle pleasures. Indeed, he was never wholly content unless he was working, and to him this was always the purpose of a holiday.

The intellectual foundations of his great career were firmly established early in life. At Morgans Academy, where he received his general education, he was an exceptionally bright pupil who surpassed his contemporaries in a wide range of subjects, which earned several medals. the first of the many he would receive in his long life. He graduated with the highest honors and went to St. Andrew's University, where he was First Foundation Scholar (1915), British Association Medallist (1915), Demonstrator in Physiology (1918), University Scholar (1919), Demonstrator of Anatomy (1920), and president of the Students' Union and Representative Council (1921). Surviving contemporaries still recall his student days and tell of hilarious and wild escapades that became legendary. Although Sir Stewart never seemed particularly amused to be reminded of these escapades, they are noteworthy as the first sparks of joyous rebellion against convention that was a facet of his character. He graduated M.A. (first class honors) in



Sir Stewart Duke-Elder.

1922 followed by M.D. (gold medal) and D.Sc. After serving as a House Officer at the Royal Infirmary, Edinburgh, he came to St. George's Hospital in London in 1923, took his F.R.C.S. in 1924 and a Ph.D. in 1925, and was appointed Honorary Consulting Ophthalmic Surgeon to St. George's Hospital and Moorfields Eye Hospital in 1928.

In 1932, when Sir Stewart was only 34, he operated on Ramsay MacDonald, then British Prime Minister, for bilateral glaucoma and he received a knighthood the following year. His fame as an ophthalmologist spread rapidly and widely; his private practice became so enormous and demanding that it is incredible he found the opportunity for any other pursuit. Yet all this time he was working on his monumental "Textbook of Ophthalmology,"

the first volume of which appeared in 1932 and the last and seventh volume in 1954. In later years this was to be followed by his even more colossal "System of Ophthalmology" in 15 volumes, begun in 1958 and completed in 1976. His other books, all of outstanding quality, appeared almost incidentally and were regularly re-edited: "Recent Advances in Ophthalmology," "Diseases of the Eye," "A Century of International Ophthalmology," "The Practice of Refraction," and the like.

It is these remarkable books on which Sir Stewart's unrivalled reputation is largely based, for he rarely taught in the ordinary didactic sense and seldom debated at congresses. Although he carried out research in his earlier days, producing papers notable for the elegance of their style and the high scientific standard of their presentation, which was far in advance of other ophthalmological publications of that time, and although he was a surgeon and clinician of repute, an editor of both the British Journal of Ophthalmology and Ophthalmic Literature, and founder of the Institute of Ophthalmology and the Faculty of Ophthalmologists, these achievements alone might not have led to the world wide acclaim he enjoyed, whereas just one of his magnificent volumes would have merited it.

All the volumes of his "Textbook," of which he was the sole author, are attractively written in a flowing style with a masterly command of the English language, and wherever the books may be opened, the reader is captured and carried along with absorbing interest. Here in these early volumes he gathered and distilled all of importance that had been written in ophthalmology in any language and in all its aspects from the earliest times. To contemplate the whole work is to find it scarcely credible that any human being could organize the material so bril-

liantly with such intellectual range, and above all, with such phenomenal industry. Here then was Sir Stewart's greatest achievement in raising the standard of ophthalmology to a distinct clinical and discipline scientific throughout the world. In so doing, he laid the foundations of modern ophthalmology, for through this medium young ophthalmologists of all nationalities emerged to surpass their predecessors not only in erudition but in knowledge of all the fascinating problems of the physiology and pathology of the eye.

No doctor in his lifetime could have been more highly revered or have received greater recognition for his achievements. Apart from his early knighthood, he was honored by being appointed Surgeon-Oculist to King George VI, King Edward VIII, and Oueen Elizabeth, and becoming a K.C.V.O. in 1949 and G.C.V.O. in 1958. In recognition for his great work as Hospitaller of the Order of St. John, he received the distinction of G.C. St.J. He held some nine honorary doctorates, six honorary fellowships, and 22 medals or prizes in ophthalmology throughout the world. He was life president of the International Council of Ophthalmology and past president of the Faculty of Ophthalmologists and the Ophthalmological Society of the United Kingdom; he was an honorary member of no less than 28 national ophthalmological societies. Probably his most prized attainments were the Gonin Medal, the highest award in international ophthalmology, and his election to the Royal Society in 1960.

Despite his great distinction and exceptional scholarship, he was never the grave, remote, tall, and forbidding figure one might have expected to meet. Quite the reverse: he was small and neat, bubbling with life, full of quips, always accessible, utterly devoid of pomposity, and

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so carefree that it was hard to believe that he had achieved so much and held such responsible positions.

The name Duke-Elder now takes its rightful place among the illustrious of medical history, and it will ever be inseparable from the annals of the Institute of Ophthalmology (London) which he founded and of which he was president when he died.

We extend our deepest sympathy to Lady Duke-Elder, also an ophthalmologist, who devoted her life to Sir Stewart in the 50 years of their marriage.

NORMAN ASHTON

Walter Sydney Atkinson, M.D. 1891–1978

Walter Sydney Atkinson, an active ophthalmologist for more than half a century, died in Watertown, New York, on Jan. 6, 1978, at the age of 86 years.

Walter Atkinson was born in Parrsboro, Nova Scotia, on Feb. 3, 1891, the second son of Martin G. and Emma Cutten Atkinson. The family moved to Mansfield, Ohio, and later to Watertown, New York, where the father established an eye, ear,



Walter S. Atkinson, M.D.

nose, and throat practice. Walter and his brother Hedley were sent to the Medical School at McGill, where Hedley graduated in 1913, Walter a year later. After an internship at the Royal Victoria Hospital in Montreal and a residency at the Herman Knapp Memorial Eye Hospital in New York, Walter joined his father and brother in practice at Watertown. Both brothers joined the Canadian Army in 1917, and after serving overseas, were discharged in 1919 and returned to the family practice. Walter became an American citizen the following year.

Atkinson belonged to many professional societies, in some of which he attained high office. He was secretary-treasurer and later president of the American Ophthalmological Society, chairman of the American Board of Ophthalmology, and vice president of the American Academy of Ophthalmology and Oto-Laryngology. He was a member of the American College of Surgeons, the American Medical Association, his state and county medical societies, the New York Commission for the blind, and the New York Academy of Medicine, and many social clubs and civic associations.

Atkinson was the author of many medical articles, and his textbook, "Anesthesia In Ophthalmology," is recognized as an authoritative work. Additionally, he served on the editorial staff of the "Quarterly Review of Ophthalmology."

Among the numerous professional honors conferred him were honorary membership in the French Ophthalmological Society, the degree of Doctor of Science from St. Lawrence University, and the Howe Medal of the American Ophthalmological Society. In 1973 the Society of Eye Surgeons, meeting in Athens, Greece, began a lecture series in his name.

He is survived by his widow, the former Mary Gamble, and by two daughters, Jeanne (Mrs. Peter H. Remington),

and Grace—"Gay"—(Mrs. W. Graham Wright). A son, John G. Atkinson, died in 1975.

Gifted and charming, a gracious host and genial companion, Walter Atkinson will be remembered with admiration and affection in those circles to which he contributed so much in so many ways.

GORDON M. BRUCE

Letters to the Editor must be typed double-spaced on $8^{1/2} \times 11$ -inch bond paper, with $1^{1/2}$ -inch margins on all four sides, and limited in length to two manuscript pages.

CORRESPONDENCE

Spectral Transmittance of Intraocular Lenses

Editor:

The findings in the excellent article, "Spectral transmittance of intraocular lenses and retinal damage from intense light sources (Am. J. Ophthalmol. 85:167, 1978), by Martin A. Mainster, further corroborate earlier work indicating that the lens serves to protect the retina from near ultraviolet light damage and provides a filter to eliminate chromatic aberration. ^{2,3} The report also emphasizes the need for replacement lenses in aphakic eyes be they spectacles, contact lenses, or intraocular lenses, which have near-ultraviolet filters. ^{3,4}

Basic research experiments that provide actual evidence of retinal damage caused by nonintense near-ultraviolet light are, unfortunately, not discussed in the Mainster report. Dogfish retina photoreceptors were observed to be damaged, both on morphological and biochemical grounds,⁵ by in vitro exposure to subsolar diffuse near-ultraviolet emitting lamps. Moreover, the photoreceptors of albino mice were totally destroyed by 12-hour

daily exposures to black light lamps between 12 to 15 months. The intensity of near-ultraviolet light again was much lower than that in sunlight. The lenses of these mice transmitted near-ultraviolet light well (little pigment present). Recently Matuk, Lou, and Parker⁷ have shown that protein synthesis in photoreceptors of rats was greatly inhibited by exposure to near-ultraviolet light. These observations point out that retinal damage from near-ultraviolet light exposure could result even from diffuse nonintense doses of near-ultraviolet light. In these cases thermal damage had not occurred. This information would then apply to human eyes without crystalline lenses.

Another situation to be considered is the use of light sensitive or light sensitizing drugs in aphakic individuals for other medically related problems, such as glaucoma or psoriasis, in which the drugs epinephrine or methoxsalen may be used. These circumstances could lead to more easily induced retinal damage than in aphakic individuals not using these drugs. Retinas of aphakic eyes could be rendered more vulnerable to nearultraviolet light exposure caused by the easier access of the light sensitive drugs to the posterior portion of the eye. An epinephrine-induced maculopathy has been reported by Kolker and Becker.8 S. G. Kramer, M.D. (personal communication) has also described an increased uptake of epinephrine by aphakic animal retinas over that in phakic controls.

Another problem of aphakic individuals with corrected vision is that of excessive glare. Vision-corrective devices with tinting would be useful in eliminating some of this glare as well as protecting the retina from damage and improving visual acuity.

I think such data suggest that all aphakic individuals be provided with replacement lenses, not only for focusing, but also for filtering near-ultraviolet light anterior to the retina.

SEYMOUR ZIGMAN, PH.D.

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Retinal Dialysis Secondary to Use of the Kaufman vitrector

Editor:

During a routine cataract extraction, vitreous appeared in the wound. The Kaufman Vitrector was inserted through a widely dilated pupil into the vitreous. After a moment of suction with a 30-ml syringe, a membrane-like structure, presumably the anterior vitreous face, entered the porthole of the Vitrector. Suddenly, a fingerlike projection of diaphanous tissue appeared from the superior periphery and approached the port of the Kaufman Vitrector. This tissue was presumably retinal and appeared to be attached to the vitreous that was being aspirated through the Vitrector. After removal of the Vitrector, excision of the vitreous with scissors and Weck sponges was performed. At the end of the procedure, the superior half of the retina floated freely in the vitreous cavity. The rotary Kaufman Vitrector is capable of winding up vitreous strands and exerting traction on the retina.

Since this complication occurred, a new model, the Kaufman Vitrector II, has been introduced. It features a reciprocating, rather than a rotary cutting head. In my opinion, the previous model should be recalled.

RICHARD L. LITWIN, M.D.

Berkeley, California

Reply

Editor:

The concept of the Vitrector I was sound and the prototype models clearly worked well. In practice, however, some of the units spooled vitreous and caused traction on the retina, which is why the original model has been replaced by the Vitrector II, a model with a smaller tip and a guillotine action that does not spool vitreous. The newer model has been exceptionally and consistently reliable in our hands. Because of the clear advantages of the Vitrector II, the company that makes these instruments has encouraged ophthalmologists to send back all models of Vitrector I and has agreed to replace them with the newer models, even though the newer model might cost slightly more. I have not had difficulties in doing several hundred vitrectomies with the newer models, which have produced far less traction than vitrectomies attempted with sponges.

HERBERT E. KAUFMAN, M.D. New Orleans, Louisiana

BOOK REVIEWS

Microsurgery of the Anterior Segment of the Eye. By Richard C. Troutman. St. Louis, C. V. Mosby Company, 1977. Hardbound, 357 pages, table of contents, index, 136 black and white figures. \$47.50

This volume describes Richard Troutman's investigations and techniques in microsurgery of the anterior segment of the eye and specifically, the cornea. Fourteen chapters cover subjects from optical considerations to instrumentation and the techniques of various keratoplasties done alone or in combination with cataract extraction, lens implantation, or glaucoma operations. Dr Troutman generally emphasizes physical disorders of the cornea and technologic management. He describes in detail the surgery of astigmatism and the continuous monitoring of corneal curvatures during operation with a keratoscope, which represent substantial advances in this field. The author's mastery of the mathematics of the optics of astigmatism and his contributions in these areas are evident.

Several controversial subjects are treated, including the use of penetrating sutures in keratoplasty or the partial suturing of the corneal graft into place before lens removal in combined operations.

The illustrations and printing are well done, but there is a great deal of white space, which might have been eliminated. The book should be in the library of all who are interested in corneal surgery. It will help not only the student or resident ophthalmologist, but also the practicing corneal surgeon.

SAMUEL D. MCPHERSON, JR.

Corneal Transplantation. By Frank M. Polack. New York, Grune and Stratton, 1977. Clothbound, 268 pages, table of contents, index, over 160 black and white photographs. \$34.50

Recent developments in instrumentation, surgical technique, donor material handling, and postoperative management have significantly improved corneal transplant results. However, many corneal surgeons have limited knowledge of the biologic and immunologic responses after keratoplasty. Similarly, laboratory investigators interested in corneal problems are often unfamiliar with the numerous clinical considerations for human corneal transplantation. Frank Polack's book contains both basic and clinical material and is directed toward those involved in corneal research and the "developing corneal surgeon."

The book is divided into four sections: (1) basic problems in tissue transplantation and the biology of the corneal graft, (2) techniques and instrumentation, (3) influence of host response in clinical results and pathology of the graft, and (4) organization and operation of the eye bank. The first section of the book gives an excellent review of the physiology and wound healing of corneal grafts, the fate of transplanted tissue, and the immunology of corneal grafts. Dr. Polack organizes previously published data with current experiments in a concise overview of the biology of corneal transplants. This section is nicely illustrated with scanning and transmission electron photomicrographs and has good references. The section on techniques and instrumentation includes a review of the currently used microsurgical instruments and sutures, and the indications and preoperative considerations for keratoplasty. The description of the author's surgical techniques for keratoplasty is necessarily brief because the reader cannot possibly learn all of the fine points of keratoplasty from a book of this scope. Some readers may disagree with the author's choice of vitrectomy instruments and the practice of usually removing the lens of keratoplasty patients over 60 years of age (p. 154). The sections on the influence of host response in clinical results and pathology of grafts, and on organization and operation of the eve bank are up-to-date and informative.

I highly recommend this book to those interested in experimental or human corneal transplantation. Although it will not teach the beginning surgeon the finer points of keratoplasty, it assembles the relevant experimental and clinical observations that are important for successful corneal transplantation.

WALTER J. STARK

Sights and Sounds in Ophthalmology, The Ocular Fundus in Neuroophthalmologic Diagnosis, vol. 3. By Neil R. Miller and Stuart L. Fine. St. Louis, C. V. Mosby Company, 1977. Clothbound, 81 pages, table of contents, preface, foreward, quizzes, answers, bibliography, index, 14 illustrations, 100 slides with accompanying cassettes (3, 2-sided). \$150

Neil Miller and Stuart Fine have compiled a truly excellent set of slides and presented many important case reports that illuminate old topics with fresh thoughts in a volume of up-to-date neuro-ophthalmology.

The slides are composites that often include explanatory illustrations, in addition to fundus photographs, x-rays, and histologic sections. The explanatory drawings are done in attractive colors and with exemplary clarity; the black and white reproductions of the drawings in the accompanying booklet pale by comparison.

The text of the booklet has been read verbatim into a tape recorder by the authors; these are the "sounds" mentioned in the title. This aural repetition of the text has some advantage. Although it might be discouraging to sit down with a slide projector and a tape recorder for three hours of concentrated listening, with the help of the booklet, one knows what to expect and may therefore proceed

out of sequence. Also, while listening, one can concentrate on the illustrations.

At the end of the presentation, a brief quiz is followed by a discussion of the correct answers, which should facilitate learning. Also included is a valuable selection of about 250 references that could serve as a starting point for the more intense study of neuro-ophthalmology.

Except for some inexplicable dark patches on a few of the visual fields (slides 17, 21, 45, and 62), this teaching set is good. Every ophthalmology, neurology, and neuro-surgery department should have at least one, so that every resident may have a chance to absorb this valuable material.

H. STANLEY THOMPSON

Interpretation of Fundus Fluorescein Angiography. By Howard Schatz, Thomas C. Burton, Lawrence A. Yannuzzi, and Maurice F. Rabb. Clothbound, 761 pages, table of contents, index, 1,142 black and white figures, 101 color figures. \$78.50

When it was first introduced in 1960, fluorescein angiography of the fundus was cumbersome and more of a laboratory curiosity than anything else. Indeed, the first paper on the subject was rejected by The IOURNAL, presumably because it was not sufficiently important. In the ensuing 18 years, the technique has become a widely accepted clinical modality, and the information resulting from its use has greatly expanded our knowledge of fundus disorders. During its infancy, fluorescein angiography was not a precise methodology, and the results did not lend themselves to a uniform intrepretation. The results, however, did lead a number of investigators to reexamine the anatomy, physiology, and pathology of the retina, pigment epithelium, and choroid with spectacular success. Our understanding of the blood-retinal and blood-pigment epithelial barriers, choroidal neovascularization, macular edema, and the various hemorrhagic and exudative fundus lesions all stem from information derived from fluorescein angiography. Today, when the technique has become standardized and routine, its value obviously depends on proper analysis.

Dozens of books and monographs and thousands of articles have been written about fluorescein angiography. This large and handsome volume, written by four of America's leading practitioners of the art and science of fundus angiography, is the first to deal primarily with the interpretation of the fundus angiograms.

An introductory section deals with general principles of the method and is prefaced by a small and charming historic chapter that includes some previously unpublished vignettes by early pioneers. Obviously, not all the major contributors to angiography could be included in such a brief history, but one would have expected some mention of Dollery and his co-workers, and of Amalric. The remainder of the first section deals with equipment, materials and methods, fundus histology, and the normal angiogram, all of which is useful. However, there is a minor error in the description of the radial peripapillary capillaries: they arise from intraretinal arterioles and not from those of the disk.

The rest of the volume is devoted to the interpretation of abnormalities seen by fluorescein angiography. For this, the authors have provided an elaborate flow sheet (a copy of which is placed in a pocket at the back of the book, but this is not mentioned in the text) that divides the entire subject into hypo- and hyperfluorescence. By following the various pathways, one will presumably make a correct diagnosis. If strictly followed, this

method should facilitate an accurate diagnosis in most instances of fundus disease that exhibits a fluorescein angiographic abnormality. Illustrative and generally excellent fluorescein angiograms are complemented by numerous histologic secprovide pseudoclinico: that pathological correlations (pseudo-only in the sense that the microscopic sections were not from the patients whose angiograms are shown, but rather are representative of the condition). Additionally, there are 101 two-color drawings that schematically illustrate many of the major fundus alterations discussed in the text. They certainly will help the neophyte understand the essence of the angiogram.

Generally, this is a useful volume for anyone interested in fundus disease. There are occasional redundancies, but they are required by the artificial division and schema used by the authors. The alternative method of teaching the subject is to list each disease and its angiographic abnormalities, which would lead to a less logical and more unwieldy text. One can imagine that by using the present schematic approach, one could design a computer program to interpret fully and accurately a fluorescein angiogram. Given the state of the art of computer technology, I dare say that it could and will be done.

PAUL HENKIND

OPHTHALMIC MINIATURE

The Prince went to vote about four in the afternoon, flanked on the right by Father Pirrone, on the left by Don Onofrio Rotolo; frowning and fair-skinned, he proceeded slowly towards the Town Hall, frequently putting up a hand to protect his eyes lest the breeze loaded with all the filth collected on its way should bring on the conjunctivitis to which he was subject; and he remarked to Father Pirrone that though the air would have been like a putrid pool without the wind, yet health-giving gusts did seem to drag up a lot of dirt with them.

Giuseppe Di Lampedusa, *The Leopard* New York, Pantheon, 1961

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

British Journal of Dermatology

PHOTOPROTECTION OF THE EYE IN PUVA THERAPY. Wennersten, G. (Dept. Dermatol., Karolinska Univ., Stockholm, Sweden). Br. J. Dermatol. 98:137, 1978.

On the basis of animal experiments it has been postulated that patients receiving the psoralens might suffer ocular damage on exposure to intense phototherapy, or even on exposure to sunlight. In fact, this has never been shown to be so for humans but nevertheless, the current approach among investigators managing PUVA treatment is to prescribe protection of the eye during therapeutic radiation and in addition, sunglasses during the rest of the day when the treatment has been given. In order to decide which sunglass would be most protective, the authors investigated the transmission of a series of sunglasses. They found that unsorted, general-purpose sunglasses were unsatisfactory since they transmitted anywhere between 16 and 67% of the ultraviolet light in the range 350 to 800 nm. On the other hand Polaroid glasses completely blocked all transmission of the ultraviolet in this range. It is interesting to note that glasses designed to darken with light intensity showed a transmission of 17% in the ultraviolet after maximal darkening. (1 figure, 1 table, 6 references)—David Shoch

British Journal of Ophthalmology

OCULAR HYPERTENSION—A LONG-TERM FOLLOW-UP OF TREATED AND UNTREATED PATIENTS. David, R., Livingston, D. G., and Luntz, M. H. (Dept. Ophthalmol., Univ. of Witwatersrand, Johannesburg, South Africa). Br. J. Ophthalmol. 61:668, 1977.

The 61 patients (117 eyes) included in this study met the criteria of: (1) repeated applanation pressure readings greater than 20 mm Hg, (2) open chamber angles, (3) not clearly glau-

comatous disks (distinct, pink-colored rims of nerve-fiber tissue all-around), and (4) normal visual fields by kinetic examination with two test objects on the Goldmann perimeter and the 1/1000 mm test object on the Bjerrum screen. The mean of three pressure readings within the first few weeks of the patient's presenting to the hospital is referred to as the mean initial intraocular pressure. Only 27 patients (50 eyes) were placed on treatment for their hypertension. The administration of pilocarpine was preceded by a careful search for, and treatment of, peripheral lattice degeneration or retinal holes. The average follow-up period, with re-examinations including perimetry every two to six months, was 42.8 months. The development of visual field defects with corresponding disk changes was observed in 12 eyes (ten patients). This happened in two of the 75 eyes with mean pressures between 21 and 25, in three of the 25 eyes with pressures between 26 and 30 mm, and in seven of the 17 eves with pressures greater than 30 mm. Treatment was of prophylactic value only insofar as it placed the eye in a lower pressure category. Treatment, on the other hand, did not seem to have any harmful effects and is considered to be indicated in elderly individuals with pressures greater than 26 mm. Younger patients are placed on treatment only if the mean pressure exceeds 30 mm Hg. (6 figures, 4 tables, 17 references)-Peter C. Kronfeld

Journal of the American Medical Association

RETINAL HEMORRHAGES IN SUBACUTE CARBON MONOXIDE POISONING. Kelley, J. S., and Sophocleus, G. J. (Dept. Ophthalmol., Greater Baltimore Med. Ctr., Baltimore, Md.). J.A.M.A. 239: 1515, 1978.

Three incidences of carbon monoxide poisoning occurred owing to defective heating systems. Twelve persons were affected; of these, three lost their lives. Because the symptoms of carbon monoxide poisoning closely resemble flu and other common illnesses, correct diagnosis was not made as promptly as it

might have been. Hemorrhages were found in the nerve fiber layer of the retina in all five of the patients who had been exposed for more than 12 hours. It is the authors' contention, therefore, that complete examination of the patient should always include ophthalmoscopy, and that the finding of retinal hemorrhages, in addition to nausea, headache, and dizziness, should alert the physician to the possibility of carbon monoxide poisoning. (4 figures, 4 references)—Authors' abstract

Journal of Pediatric Ophthalmology

VISUAL ACUITY AND BINOCULARITY IN CHILDREN WITH UNILATERAL ACQUIRED APHAKIA. Frank, J. W., and France, T. D. (Dept. Ophthalmol., Univ. of Wisconsin, Madison, Wis.). J. Pediatr. Ophthalmol. 14:200, 1978.

Seventeen cases of unilateral aphakia secondary to acquired cataracts and dislocated lenses were presented. Final visual acuity was 6/22 (20/70) or better in 12 of our 17 patients. Binocular single vision has been maintained in four cases. Ten patients had a posttherapy tropia.

Early surgery and treatment particularly in the young child is stressed in order to maintain normal binocular single vision. Where once traumatized eyes were felt to be "sick" eyes and better left alone, current surgical and therapeutic methods make restoration of vision and binocularity a reasonable goal. (3 tables, 15 references)—Authors' abstract

SURGICAL CORRECTION OF DISSOCIATED VERTICAL DEVIATIONS. Braverman, D. E., and Scott, W. E. (Dept. Ophthalmol., Univ. of Iowa Hosp., Iowa City, Ia.). J. Pediatr. Ophthalmol. 14:337, 1978.

The surgical treatment and results of 17 patients with dissociated vertical deviations is presented. Supermaximum recession of the superior rectus muscle was performed on the deviating eye. No lid changes or any limitation of elevation were noted following the surgery. The manifest deviation was converted to less than ten prism diopters in all except one case. Four over-corrections of two to six prism diopters of hypotropia resulted. There was no

change in eye preference to the operated eye. Saccadic velocities showed no change in the acceleration or velocity of the operated superior rectus. (1 figure, 3 tables, 17 references)—Authors' abstract

Klinische Monatsblätter für Augenheilkunde

LONG-ACTING LOCAL ANESTHETICS—AN ENRICHMENT IN OPHTHALMIC SURGERY? (German) Schlegl, H. J. (Univ. of Saarland, Hamburg, West Germany). Klin. Monatsbl. Augenheilkd. 171:359, 1977.

Bupivacain, a long-acting local anesthetic, was used in 300 eye operations. Of the .25%, .5% and .75% concentrations, .5% was found reliable. The safe dose is up to 30 ml or 150 mg (2 mg/kg). The onset of good anesthetic effect was occasionally as early as ten minutes, but usually 30 minutes after injection. One advantage of this waiting period was that any swelling or pressure from the injection disappeared. Good akinesia and anesthesia was present in all patients for at least two hours and probably longer, although the exact duration of the effect was not determined. Ninety-one percent of operated patients had no pain the evening or morning after surgery. (4 figures, 1 table, 36 references)-Peter Egbert

EXUDATIVE SENILE MACULOPATHY. CLINICAL PICTURE, PATHOGENESIS, PROGNOSIS AND THERAPY. (German) Wessing, A. (Ederhard-Karls Univ., Tübingen, West Germany). Klin. Monatsbl. Augenheilkd. 171:371, 1977.

The natural history of exudative senile macular degeneration was observed in 65 eyes. At the end of a 19-month observation period, 4% of the eyes had better vision, 48% were unchanged, and 48% had worse vision. Twentyseven eyes with similar maculopathy were treated by photocoagulation and followed for 20 months. The overall visual results of these treated eyes were no better than the untreated control eyes. However, out of the large category of exudative senile maculopathies, there are infrequent special situations that may benefit from photocoagulation treatment: (1) an uncomplicated detachment of the retinal pigment epithelium without a subretinal vascular network, (2) a beginning, localized subretinal vascular network, and (3) certain late cases with regressing vascularity in which it is possible to hasten final scarring and limit the scotoma. The authors feel their results are less optimistic than those of others because of a longer follow-up. They emphasize that an immediate improvement in vision after photocoagulation all too often later disappears. (15 figures, 32 references)—Peter Egbert

PRIMARY EPIRETINAL GLIOSIS. (German) Spitznas, M., and Leuenberger, R. (Univ. Eye Clinic, Essen, West Germany). Klin. Monatsbl. Augenheilkd. 171: 410, 1977.

Primary epiretinal gliosis, also called macular pucker, cellophane maculopathy, surface wrinkling retinopathy and idiopathic preretinal macular fibrosis, occurs in otherwise healthy eyes. Forty-five patients (51 eyes) with this condition were studied. Most frequently, the foveal region was involved but the foveola was often spared. With increased distance from the center of the macula, the frequency of involvement decreased. The density of the membrane varied-22 were transparent, 20 translucent gray and nine opaque white. The average visual acuity was 6/15 (20/50); in no case was it worse than 6/122 (20/400). In one patient, the membrane spontaneously detached from the retina into the vitreous and the vision improved. The incidence of the disease was greater in both myopia and hyperopia than in emmetropia. The average age of the patients was 55 years (range 8 to 77) with no sex predilection. (3 figures, 9 tables, 38 references)-Peter Egbert

FACTORS INFLUENCING THE URGENCY OF REPAIR OF RETINAL DETACHMENT. (German) Kreissig, I. (Eye Clinic, Univ. Bonn, Bonn, West Germany). Klin. Monatsbl. Augenheilkd. 171:530, 1977.

In order to determine preoperative factors which influence visual results, the visual acuity of 266 patients was determined for one year after retinal detachment surgery. Preoperative factors which were associated with a poor final visual acuity were a detachment which included a total macular detachment, macular detachment for greater than one week, and increasing age. A partially detached macula recovered good vision—equivalent to a detachment which did not involve the macula. A macula that was detached for two weeks re-

covered significantly less vision than one detached only one week. However, there was little difference between a detachment of two weeks and greater than two weeks. Patients over 55 years old had vision equal to younger patients two months after surgery, but after two months the younger patients continued to improve and the older ones did not. Therefore, after one year the vision was significantly better in the younger patients.

The author feels detachment surgery should be performed without delay when: the macula is attached but threatened by an approaching bullous detachment; the macula is partially detached; the macula has been detached for less than one week; or the macula is detached in an elderly patient. (10 figures, 5 references)—Peter Egbert

AQUEOUS HUMOR LEVELS OF GENTAMICIN IN MAN AFTER PARENTERAL SUBCONJUNCTIVAL AND TOPICAL ADMINISTRATION. (German) Ultermann, D., Matz, K., and Meyer, K. (Depts. of Ophthalmol. and Bacteriology, St. George Hosp., Hamburg, West Germany). Klin. Monatsbl. Augenheilkd. 171: 579, 1977.

Varied amounts of gentamicin were given to 135 patients before cataract extraction. Aqueous humor samples were withdrawn at the start of surgery to determine ocular penetration of the drug. After intravenous injection of 80 mg of gentamicin, an aqueous level of only 0.5 mcg/ml was reached. This is not a therapeutic level for most bacteria. Subconjunctival injection of 40 mg of gentamicin consistently gave aqueous concentration greater than 10 mcg/ml within 15 minutes. This level was maintained for 12 hours, and then fell between 12 and 24 hours after injection. Subconjunctival injection of 20 mg also usually achieved therapeutic aqueous concentrations but 10 mg did not. Topical application did not give therapeutic aqueous levels. For intraocular infection, 40 mg of gentamicin should be given subconjunctivally twice a day. (2 figures, 8 references)—Peter Egbert

New England Journal of Medicine

CRYPTOCOCCAL ENDOPHTHALMITIS AFTER CORNEAL TRANSPLANTATION.

Beyt, B., Jr., and Waltman, S. R. (Dept. Med., Washington Univ. School of Med., St. Louis, Mo.). N. Engl. J. Med. 298:825, 1978.

A cornea was obtained from a 25-year-old donor who died due to progressive respiratory failure secondary to pneumonitis, after a three-year history of polymyositis treated with corticosteroids and intermittent cyclophosphamide. Two months after the graft was performed, examination revealed decreased visual acuity and a yellow-white mass in the anterior chamber. Aqueous aspirated from the anterior chamber grew Cryptococcus neoformans. Cultures of blood drawn 11 days before the death of the donor, eventually grew the same organism. With the recognition that occult opportunistic infections may be transmitted by corneal graft, immunosuppressed patients with known opportunistic infections should be excluded as corneal donors for they may have multiple, often unrecognized infections. (1 figure, 5 references)—David Shoch

Ophthalmologica

ULTRASTRUCTURAL OBSERVATIONS ON THE RETINA IN TYPE II GLYCOGENOSIS (POMPE'S DISEASE). Goebel, H. H., Kohlschütter, A. and Pilz, H. (Division of Neuropathology, Univ. of Göttingen, Göttingen, West Germany). Ophthalmologica 176:61, 1978.

The retina of a nine-month-old boy afflicted with biochemically proven type II glycogenosis contained abundant lysosomal glycogen. This was present in almost every cell type and occasionally associated with lipofuscin in choroidal macrophages. Lysosomal glycogen was absent from melanocytes and pigment epithelial cells. No degeneration of any cell layer was noted. The ubiquitous accretion of lysosomal glycogen resembles the widespread distribution of lipopigments in canine neuronal ceroid lipofuscinosis, another lysosomal disorder. (6 figures, 16 references)—Authors' abstract

Pediatrics

ELECTRORETINOGRAPHY IN NEONATES TREATED WITH PHOTOTHERAPY. Bhu-

pathy, K., Sethupathy, R., Pildes, R.S., Constantaras, A.A., and Fournier, J.H. (Divisions of Neonatology and Ophthalmol., Univ. of Illinois College Med., Chicago, Ill.). Pediatrics 61:189, 1978.

Electroretinographic (ERG) studies were performed in 22 normal control newborns and 28 neonates who had had phototherapy during the first few days of life. Mean age at ERG testing was 16.8 ± 2.9 days in the phototherapy group and 16.4 ± 3.7 days in the control group. Results of routine fundoscopy were normal in both groups. Amplitudes of a and b waves under dark- and light-adapted states were similar in both groups. Phototherapy did not appear to have deleterious effects on photopic and scotopic retinal function in infants whose eyes were adequately shielded (2 tables, 12 references)—Authors' abstract

Science

GYRATE ATROPHY OF THE RETINA: INBORN ERROR OF L-ORNITHINE:2-OXOACID AMINOTRANSFERASE. O'Donnell, J. J., Sandman, R. P., and Martin, S. R. (Dept. of Ophthalmol., Univ. of California, San Francisco, Calif.). Science 200:200, 1978.

One of the few hereditary retinal degenerations that has been associated with a metabolic defect is gyrate atrophy in which elevated levels of blood and urine ornithine have been demonstrated. In this paper the authors demonstrate that the cultured fibroblasts from a patient with gyrate atrophy of the retina do not convert L-ornithine uniformly labeled with carbon-14, to proline. This metabolic block is caused by deficient L-ornithine:2-oxoacid aminotransferase activity. The patient's heterozygote father had intermediate activity of this enzyme. (2 figures, 8 references)—David Shoch

Transactions of the Ophthalmological Societies of the United Kingdom

VALUE OF PROPHYLACTIC PERIPHERAL IRIDECTOMY ON THE SECOND EYE IN

ANGLE-CLOSURE GLAUCOMA. Snow, J. T. (Canterbury, England). Trans. Ophthalmol. Soc. U.K. 97:189, 1977.

The records of a group of 72 patients who had acute angle closure in one eye and no treatment in the second were reviewed. Of these 72 patients, 40% had no trouble in the second eye but in 60% the second eye was affected and of this 60% comprising 43 eyes, 32 had either acute or chronic angle closure glaucoma in the second unoperated eye. The author compares this with a previous series of 63 patients who had prophylactic peripheral iridectomy and concludes that the likelihood of complications and eye problems is greater in those patients who do not have prophylactic peripheral iridectomies in the second eye after angle closure glaucoma in the first. (6 tables, 10 references)—David Shoch

CONSERVATIVE MANAGEMENT OF CENTRAL SEROUS RETINOPATHY. Lyons, D. E. (Taunton, England). Trans. Ophthalmol. Soc. U.K. 97:214, 1977.

A study of 26 patients (30 attacks) with central serous retinopathy was made in order to assess the result of conservative management, and the factors which might affect the

incidence of residual symptoms. There were two groups, one given systemic corticosteroids and one given no treatment. Comparison is made with two groups, one treated by laser coagulation and another given no treatment. There was no permanent serious visual loss in any patient. The final visual acuity was 6/6 (20/20) or better in 76% of the eyes, 6/9 (20/30)in 20%, and 6/12 (20/40) in 3%. There was no relationship between the final visual acuity and the duration of the attack, nor was there any relationship between the age of the patient and the duration of the attack. The treated group did no better in terms of the final visual acuity than the untreated group. The author compares his results with figures in the literature on patients treated with the laser. He notes that in the laser-treated patients, about 60% achieved 6/6 (20/20) vision or better, while in his untreated group 76% achieved 6/6 (20/20) or better vision. Finally no specific factor (the presenting visual acuity, the worst visual acuity, the duration of the attack, or the age of the patient) can be used to determine which patients should be advised to undergo photocoagulation. However, residual symptoms were more common in this series in cases in which the central serous retinopathy had lasted for three months or more. (2 figures, 4 tables, 6 references)—David Shoch

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

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For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least three months before the date of occurrence.

INTERNATIONAL CONGRESS IN OPHTHALMOLOGY: NEW DEVELOPMENTS IN OPHTHALMOLOGY

An international congress in ophthalmology will be held in Nijmegen, Holland, Sept. 25 and 26, 1978. There will be lectures on vitreous body, retina, and cornea surgery and lens implantation. For further information, write A. F. Deutman, M.D., Department of Ophthalmology, Sint Radboudziekenhuis, Philips van Leydenlaan, Nijmegen, The Netherlands.

INSTITUTO BARRAQUER: VIII INTERNATIONAL COURSE

The VIII International Course of the Instituto Barraquer will be held May 16-21, 1982. Further details will be announced later.

EYE BANK ASSOCIATION OF AMERICA, INC.: SCIENTIFIC PROGRAM

The Eye-Bank Association of America will hold a scientific program on Saturday, Oct. 21, 1978, from 9:00 A.M. to 12:30 P.M. at the Radisson Muehlebach Hotel, Kansas City, Missouri, preceding the American Academy of Ophthalmology meeting. Papers relating to corneal transplantation and eye bank techniques are solicited. Send abstracts before Sept. 8, 1978, to: R. D. Richards, M.D., Department of Ophthalmology, University of Maryland Hospital, Baltimore, MD 21201.

MIDWEST GLAUCOMA MEETING AND ASBURY LECTURE

The University of Cincinnati's Department of Ophthalmology and Alumni Association and Cincinnati Society of Ophthalmologists and CONMED will sponsor the Midwest Glaucoma Meeting and Asbury Lecture Nov. 9–11, 1978, at the Netherland Hilton Hotel, Cincinnati, Ohio. The fee is \$150 (residents and fellows, \$50). For further information, call CONMED 513-872-5486.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY: NEW OFFICERS

The following members of the Minnesota Academy of Ophthalmology and Otolaryngology took office at the May 1978 meeting of the Academy in

Duluth, Minnesota: Theodore M. Gill, president; Melvin Sigel, president-elect, otolaryngology; Harry S. Friedman, president-elect, ophthalmology; and James P. Dunn, secretary-treasurer.

ST. LUKE'S HOSPITAL: COURSE ON CLINICAL MANAGEMENT OF RETINAL DISEASE

St. Luke's Hospital, Cleveland, Ohio, announces a course "Clinical Management of Retinal Disease: A Review of Current Practical Evaluation and Treatment of Retinal Diseases," June 8 and 9, 1978. The registration fee is \$250 and \$35 for optional tapes (nonresidents, \$75). For further information, write L. Singerman, M.D., 11201 Shaker Blvd., Cleveland, OH 44104.

THIRD ANNUAL OPHTHALMIC SURGERY SEMINAR, 1978: MODERN TECHNIQUES IN MICROSURGERY AND STRABISMUS SURGERY

A two-day course of Modern Techniques in Microsurgery and Strabismus Surgery will be offered on August 11 and 12, 1978, in Rochester, New York. For further information, write Joseph D. Silverberg, M.D., Seminar Chairman, Department of Ophthalmology, Park Ridge Hospital, 1555 Long Pond Rd., Rochester, NY 14626.

MOORFIELDS EYE HOSPITAL: MICROSURGERY COURSE

The Department of Clinical Ophthalmology, Moorfields Eye Hospital, will conduct a course in Ophthalmic Microsurgery Oct. 25–27, 1978. This will be a practical course on common intraocular surgical procedures. For further information, write Mrs. J. F. Field, Microsurgical Course Secretary, Department of Clinical Ophthalmology, Moorfields Eye Hospital, City Road, London, ECIV 2PD, England.

UTAH OPHTHALMOLOGICAL SOCIETY: SUMMER SHAKESPEAREAN FESTIVAL MEETING

The Summer Shakespearean Festival Meeting will take place Aug. 4 and 5, 1978, in Cedar City,

Utah. The subject will be external diseases. For further information, write William A. Bohart, M.D., Secretary, Utah Ophthalmological Society, 960 E. First South, Salt Lake City, UT 84102.

INTERNATIONAL GLAUCOMA CONGRESS III

. The joint meeting of International Glaucoma Congress III and the 14th Annual Scientific Assembly of the American Society of Contemporary Ophthalmology will be held Jan. 14–19, 1979, at Caesar's Palace in Las Vegas, Nevada. For further information, write Martin Szanto, M.D., Suite 1110, 6 N. Michigan Ave., Chicago, Il 60602.

Universita degli Studi, Milano: International Symposium on Pituitary Microadenomas

The Università degli Studi, Milano, will hold an International Symposium on Pituitary Microadenomas in Milan, Italy, Oct. 12–14, 1978. The program will include 20 to 22 invited lectures, two open discussions on Problems in pathogenesis of pituitary microadenomas and Problems in treatment of pituitary microadenomas, and about 30 free communications. The official language will be English, and the proceedings, including lectures, selected communications, and discussions will be published by Academic Press in the Serono Symposia series. For further information, write the Secretariat of the International Symposium on Pituitary Microadeno-

mas, Clinica Neurochirurgica dell'Università, Ospedale Policlinico, Pad. Beretta, via F. Sforza 35, 20122 Milano, Italy.

PERSONALS

THOMAS D. DUANE

Thomas D. Duane has been named to serve as a member of the National Advisory Eye Council of the National Institutes of Health.

Dr. Duane is ophthalmologist-in-chief of Wills Eye Hospital in Philadelphia, as well as Professor and Chairman, Department of Ophthalmology, Jefferson Medical College.

THOMAS J. KIRBY

Thomas J. Kirby assumed office as president of the Joint Commission on Allied Health Personnel in Ophthalmology on Jan. 1, 1978. Dr. Kirby is consultant in ophthalmology at the Mayo Clinic; associate professor of ophthalmology at the Mayo Medical School.

ROBERT W. RODIECK

Robert W. Rodieck has been named Bishop Professor of Ophthalmology at the University of Washington, a chair endowed by the Bishop Foundation of Seattle. Dr. Rodieck has served most recently as Reader in Physiology at the University of Sydney.

VISUAL FIELD CHARTS

Central and peripheral field charts are available without charge to authors who require them to illustrate their papers. Write to Editorial Correspondent, American Journal of Ophthalmology, 233 East Ontario Street, Suite 1401, Chicago, Illinois 60611.

INSTRUCTIONS TO AUTHORS

For the preparation of manuscripts for American Journal of Ophthalmology

THE AMERICAN JOURNAL OF OPH-THALMOLOGY publishes original and timely contributions dealing with clinical and basic ophthalmology. Each article submitted is evaluated by two or more referees who recommend that the paper be (1) accepted unchanged, (2) returned for revision and subsequent editorial consideration, or (3) rejected. Acceptance is conditioned by such factors as the originality, significance, and soundness of the contribution; the suitability of the subject matter for subscribers of THE JOURNAL; and, the care with which the manuscript has been prepared.

Papers are accepted on the condition that they have not been published or accepted for publication in any other journal, whether printed in English or any other language. On occasion, a paper read before a society and published in the society's transactions will be considered if the society publication does not reach as wide an audience as the contribution merits. When submitting such a paper,

the author must indicate the time and place of the meeting and the name of the society publication. It is not possible to coordinate the date of publication of such papers in THE JOURNAL with that in the society publication.

Authors will be advised promptly of receipt of their papers. Thereafter they will be advised within 30 days of acceptance, rejection, or need for revision. Manuscripts that require extensive editorial correction or retyping will be returned for that purpose.

MECHANICAL PREPARATION OF MANUSCRIPT The manuscript should be prepared in the style used by THE JOURNAL. A heavy grade of white bond paper measuring 8½ by 11 inches should be used. Margins should be at least 1½ inches on all sides. Paragraphs should be indented at least one-half inch. Two copies must be submitted; carbon copies and machine-duplicate copies are acceptable only as second copies.

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The entire manuscript should be double- or triple-spaced. Single-spacing should not be used anywhere in the manuscript. The entire manuscript—including title page and footnotes, text, acknowledgments, references, tables, and legends—should be double-spaced. Rules and underlining should not be used anywhere in the manuscript.

The manuscript should be arranged in the following order:

- 1. Title page (with footnotes)
- 2. Text and summary
- 3. Acknowledgments
- 4. References
- 5. Tables
- 6. Legends for figures

Each major section should begin on a separate sheet.

TITLE PAGE The title page should be numbered page 1 and contain the running (abbreviated) heading, the title, each author's name and highest degree, and the city and state where the work was carried out. The institution and the organization(s) sponsoring the study should be credited in a footnote. A second footnote should give the name and mailing address of the author to whom correspondence should be directed. Each page after the title page should show the page number, senior author's name, and running title in the upper righthand corner.

ORGANIZATION OF CONTENT Manuscripts should be carefully organized and prepared in the style used by THE JOURNAL. A statement of the problem should be presented first. The material and methods should then be precisely described. All techniques used should be described completely enough to permit the reader to duplicate the study.

Following a description of material and methods, the results of the study should be given. A section devoted to discussion should follow. The discussion should relate directly to the topic of the paper.

Summary—Each paper must have a summary that describes the content of the paper in no more than 150 words. The author should state precisely what was accomplished, and avoid generalities.

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style is used by THE JOURNAL for periodicals (1) and for books (2):

- 1. Terry, T. L.: Extreme prematurity and fibroblastic overgrowth of persistent vascular sheath behind each crystalline lens. Am. J. Ophthalmol. 25:203, 1942.
- 2. Reese, A. B.: Tumors of the Eye, 2nd ed. New York, Hoeber, 1964, p. 91.

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TABLES Each table must be titled, numbered with an arabic numeral, and cited in the text. The title should appear directly below the designation, Table 1, and well below the title, a double rule (two parallel lines) should span the width of the table. Between this double rule and a similar, full-width single rule, a heading must be given for each column. Smaller rules spanning several columns can be used to separate main (several-column) headings from subheadings, if necessary. No other vertical or horizontal lines should be used. Each table should be double-spaced, and nothing in the table should be underscored.

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Fig. 1 (Jones, Smith, and Brown). Histologic section of the eye (hematoxylin and eosin, ×70).

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Instructions to Authors—CONT.

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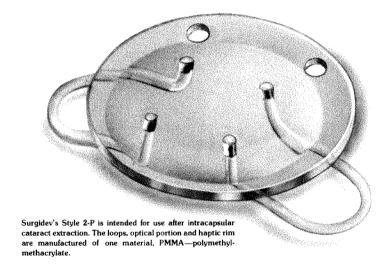
Council of Biology Editors, Committee on Form and Style: CBE Style Manual, 3rd ed. Washington, D.C., American Institute of Biological Sciences, 1972.

A Manual of Style, 12th ed. Chicago, University of Chicago Press, 1969.

Strunk, W., Jr., and White, E. B.: The Elements of Style, 2nd ed. New York, Macmillan Co., 1972.

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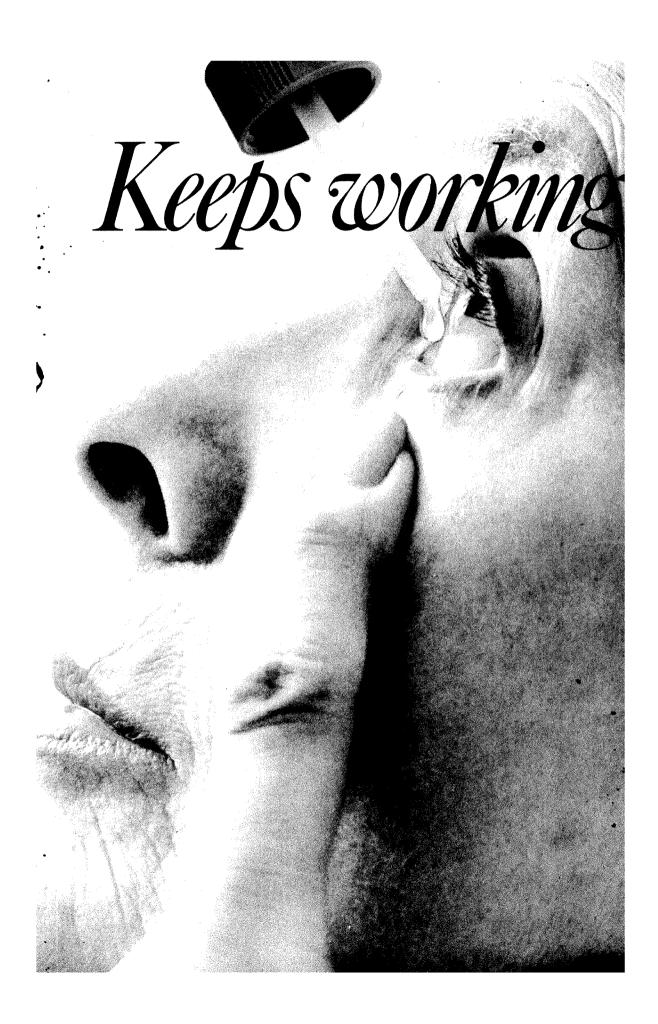
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Precautions: 1: Goniescopy is recommended prior to initiation of therapy.

2. Where there is a guiescent uveitis or a history of this condition, anticholinesterase therapy should be avoided or used cautiously because of the intense and persistent miosis and ciliary muscle contraction that may occur.

3. While systemic effects are infrequent, proper use of the drug requires digital compression of the nasolacrimal ducts for a minute or two following instillation to minimize drainage into the nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation.

should be washed immediately following instillation.

4. Temporary discontinuance of medication is necessary if salivation, urinary incontinence, diarrhea, profuse sweating, muscle weakness, respiratory difficulties, or cardiac irregularities.

Sequents receiving PHOSPHOLINE IODIDE who are exposed to carbamate or organophosphate type insecticides and pesticides (professional gardeners, farmers, workers in plants manufacturing or formulating such products, etc.) should be warned of the additive systemic effects possible from absorption of the pesticide through the respiratory tract or skin During periods of exposure to such pesticides, the wearing of respiratory masks, and frequent washing and clothing changes may be advisable. advisable

6. Anticholinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulcer pronounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism and other disorders that may respond adversely to vagotonic effects.

7. Anticholinesterase drugs should be employed prior to ophthalmic surgery only as a considered risk because of the possible occurrence of hyphema.

8. PHOSPHOLINE (ODIDE) (echothiophate iodide) should be used with great caution, if at all, where there is a prior history of retinal detachment.

Adverse Reactions: 1. Although the relationship, if any, of retinal detachment to the administration of PHOSPHOLINE (DDIDE has not been established, retinal detachment has been reported in a few cases during the use of PHOSPHOLINE (DDIDE has not been established, retinal detachment has been reported in a few cases during the use of PHOSPHOLINE (DDIDE in adult patients without a previous history of this disorder.

2. Singing, burning, lacinmation, lid muscle twitching, conjunctival and citiary redness, browache, induced myopia with visual blurring may occur.

3. Activation of latent irits or uveitis may occus.

4. Iris cysts may form, and if treatment is continued, may enlarge and obscure vision. This occurrence is more frequent in children. The cysts usually shrink upon discontinuance of the medication, reduction in strength of the drops or frequency of instillation. Parely, they may rupture or break free into the aqueous. Regular examinations are advisable when the drug is being prescribed for the treatment of accommodative sostropia.

5. Prolonged use may cause conjunctival thickening, obstruction of nasolacrimal canals.

6. Lens opacities occurring in patients under treatment for glaucoma with PHOSPHOLINE IODIDE have been reported.

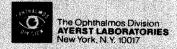
5. Prolonged use may cause conjunctival mickening, dostruction of nasolacrimal carials.

6. Lens opacities occurring in patients under treatment for glaucome with PHOSPHOLINE IODIDE have been reported and similar changes have been produced experimentally in normal monkeys. Routine examinations should accompany clinical use of the drug.

7. Paradoxical increase in intraocular pressure may follow anticholinesterase institlation. This may be alleviated by prescribing a sympathornimetic mydriaticsuch as princylephrine.

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SYMPOSIUM ON THE MANAGEMENT OF COMPLICATIONS ASSOCIATED WITH THE VARIOUS INTRAOCULAR LENSES

Jack Hartstein, M.D., Moderator

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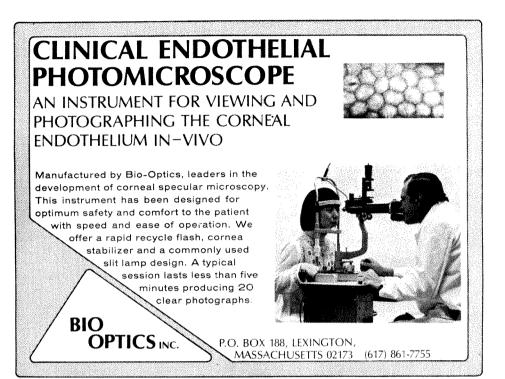
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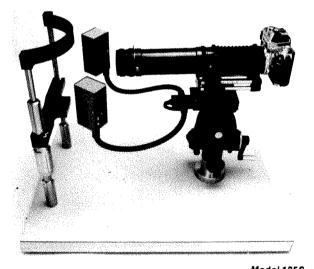
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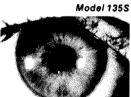
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PRELIMINARY PROGRAM

Monday, January 15: INTERNATIONAL GLAUCOMA CONGRESS III. Faculty: Drs. Mansour Armaly, Washington, D.C.; Hugh Beckman, Detroit; Paul Bregeat, Paris, France; J. Draeger, Bremen, Germany; Jules Francois, Ghent, Belgium; John Fronimopoulos, Athens, Greece; Edward Grom, Caracas, Venezuela; Fritz Hollwich, Munster, Germany; Yoshiaki Kitazawa, Tokyo, Japan; Nicholas Lambrou, Athens, Greece; Maurice Langham, Baltimore; W. Leydhecker, Wurzburg, Germany; Bernard Schwartz, Boston; Dong Shin, Detroit; Ernest G. A. van Beuningen, Frankfurt, Germany; Jacob Wilensky, Chicago.

Tuesday, January 16: INTERNATIONAL GLAUCOMA CONGRESS III (continued). Faculty: Drs. Manracuity: bits in Exnational Glaucoma Congress III (continued). Facuity: bits. mansour Armaly, Washington, D.C.; Jose Barraquer, Bogota, Colombia; John Beale, Jr., San Francisco; Ido Egerer, Vienna, Austria; Dieter Friedburg, Dusseldorf, Germany; Miles Galin, New York; H. Harms, Tubingen, Germany; Irving Katz, West Point, Pennsylvania; G. K. Krieglstein, Wurzburg, Germany; Maurice Langham, Baltimore; W. Leydhecker, Wurzburg, Germany; Stephen Obstbaum, New York; Bernard Schwartz, Boston; G. Scuderi, Rome, Italy; H. Saul Sugar, Detroit; J. Wollensak, Berlin, Germany; Thom Zimmerman, New Orleans.

Complete program includes:
Wednesday, January 17: INTRAOCULAR LENS/CATARACT SURGERY SEMINAR Chairman:
Richard C. Troutman MD, State University of New York, Brooklyn. Speakers: John P. Beale,
Jr. MD, Jules Francois MD, Jack Hartstein MD, Richard Kratz MD, Harold Scheie MD,
Edward Shaw MD, John Shock MD. Other speakers to be announced.

Thursday, January 18: CORNEA AND EXTERNAL DISEASES SEMINAR Chairman: Herbert E. Kaufman MD, Louisiana State University, New Orleans. Speakers to be announced. CONTACT LENS SEMINAR Chairman: Antonio R. Gasset MD, University of Florida, Gainesville. Speakers to be announced.

Friday, January 19: VITREOUS/CHOROID/RETINA SEMINAR Chairman: Harvey Lincoff MD, Cornell University, New York. Speakers to be announced.

The program on Sunday, January 14, will feature a special COSMETIC SURGERY SEMINAR to be held under the chairmanship of Pierre Guibor MD, New York.

Program also will include afternoon tutorials and workshops on Ophthalmic Microsurgery, Intraocular Lens Surgery, Ultrasonography, Strabismus, External Ocular Diseases, Metabolic Eye Diseases, Anterior Segment Diseases, The Lacrimal System, Gonioscopy, Dyslexia and Perceptual Problems in Ophthalmology, Beta-Adrenergic Blocking Agents in Glaucoma, and many others.

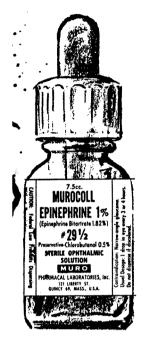
CME CREDIT: As an organization accredited for continuing medical education, the American Society of Contemporary Ophthalmology certifies that this continuing medical education activity meets the criteria for 40 credit hours in Category 1 of the Physician's Recognition Award of the American Medical Association. The program also meets the criteria for 40 hours of CME credit in Category 1 for the Certificate of Advanced Medical Studies in Ophthalmology of the American Society of Contemporary Ophthalmology.

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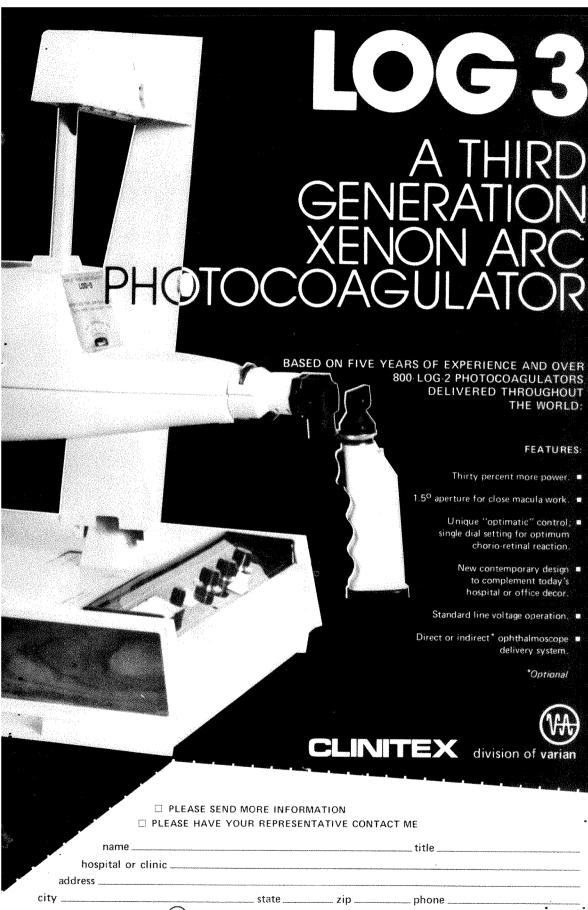
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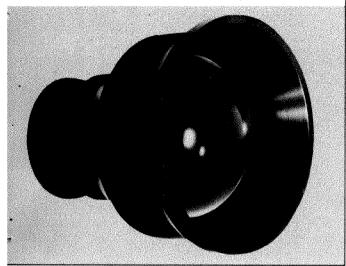
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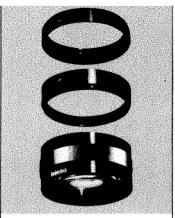
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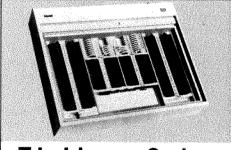
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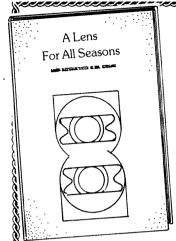
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Guest Faculty:

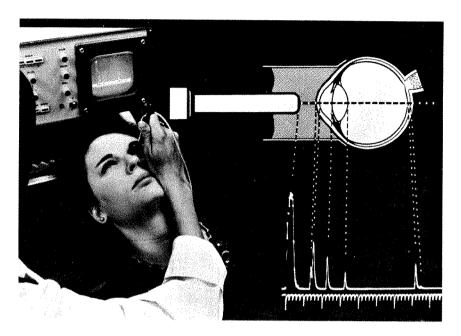
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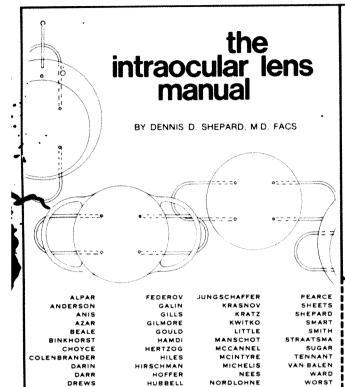
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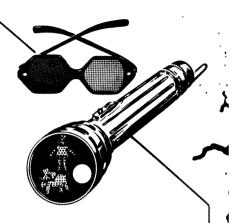
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British Journal of Ophthalmology March 1978 Vol. 62 No. 3

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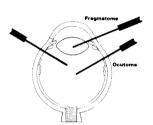
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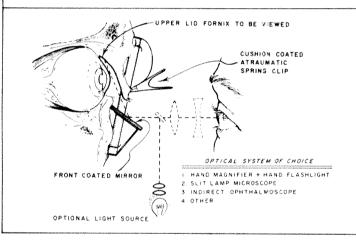
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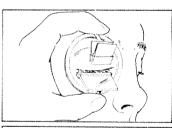
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